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INTESTINAL OBSTRUCTION IN CHILDREN

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THE mortality of intestinal obstruction is still so high and the early recognition of the disease is so important that the writers believe frequent consideration of the subject, by the society, should be of value. This paper is based on a study of one hundred and five cases of obstruction occurring in one hundred and two patients operated on since 1915 by me and my associate, Dr. George D. Cutler.

Intussusception is, by far, the commonest cause of obstruction in childhood but a few cases of obstruction from other causes are included in this paper to give a little broader point of view of the subject. In this series there were eighty-eight cases of intussusception and seventeen cases of obstruction from other causes. This proportion is very different from that found in the adult general hospital. Dr. E. P. Richardson[1] in a recent paper states that in one hundred and eighteen cases of obstruction occurring in the Massachusetts General Hospital in ten years, there were twenty cases of intussusception and ninety-eight from other causes.

In our series of intussusception, fifty-six cases occurred in males and thirty-two in females, which is the common predominance found by other observers. The age is of importance. The youngest patient was three weeks old; the oldest eight years and eleven months. Sixty-six patients were under one year of age. Ten cases occurred during the second year, while twelve occurred between the second and ninth years.

The commonest type of child to have intussusception is the fat, healthy-looking male baby in its first year of life.

In this series two cases of intussusception have occurred as a sequel to appendectomy; one before the patient was discharged from the hospital and one two or three days after his discharge from the hospital. One case occurred during a diarrhoea and one followed purpura. Such occurrenes, however, are the exception, not the rule.

It is unnecessary to spend time with a description of the clinical manifestations of this disease as they have been well and accurately described in numerous publications[2] and should be familiar to all. It is still true, however, that in this community patients frequently reach the surgeons too late. The causes of this are: (1) Failure of parents to recognize the importance of a "belly-ache" in their child and delay in calling the doctor until they become exercised by the appearance of blood in the stool. (2) Failure of the physician to make a careful abdominal examination. It is still not very uncommon to have a parent tell us that their doctor ordered castor oil over the telephone without seeing the patient. Every case of abdominal pain and vomiting demands a careful abdominal and rectal examination. The diagnosis is occasionally but usually not difficult.

In patients in whom the invaginated gut had not become so swollen and edematous that it could not be reduced, the mortality was high but not excessive. In our series there were seventy-two such patients operated on; fourteen of them died and fifty-eight recovered, a mortality of nineteen and four tenths percent.

Of the remaining patients on whom resection or enterostomy was performed the mortality was very high. In nine cases in which resection and lateral anastomosis was performed eight proved fatal. The one recovery is, perhaps, worthy of note. This took place in a child of three who had a double intussusception af the ileo-ileo-colic variety. The small bowel was reduced from the colon without undue difficulty. The intussusception of the ileum could not be reduced. About a foot of it was resected and a lateral anastomosis performed. Four days later the stoma, even though a large one, had failed to work. The patient had fecal vomiting, visible peristalsis, distention and was rapidly failing. An ileostomy was performed which proved to be a life saving procedure. Four months later this ileostomy was closed without incident. The child was seen two years later in excellent health and having complained of no abdominal or digestive difficulties.

Two of the resections were performed according to the technique recommended by Mr. Jesset[3]; both were unsuccessful. In three patients enterostomy was performed following reduction in the hope of lessening the toxemia. In one case the patient had much less reaction and rise of temperature than would have been expected from the operative findings and recovered. The other two developed pneumonia and died. Two patients had temporary enterostomy performed to relieve excessive distention; both proved fatal.

The seventeen cases of obstructon due to causes other than intussusception are as follows: Three of these cases were subsequent to intussusception operations. In one a band had formed across the ileum almost at the ileo-cecal valve actually sloughing through about one-half its diameter. In this case a cecostomy was performed, the ileum drawn through the ileo-cecal valve, a glass tube tied into its end and the cecostomy closed to the tube. The child recovered and the cecostomy was later closed. This is a much quicker and safer procedure than lateral anastomosis and more desirable and, we believe, safer than simple ileostomy which would later require anastomosis. One other case of obstruction following intussusception was caused by a band across the ileum originating in a caseous mesenteric gland. This child recovered after the band had been freed. The third case was caused by a band and complicated by mesenteric thrombosis and the patient died.

Two cases occurred as a result of Meckel's diverticulum becoming adherent across the ileum; one recovered and one died. Three cases had obstruction during the first ten days of convalescence following operation for appendicitis with peritonitis. Ileostomy was performed for the relief of this condition in two patients who recovered. It is our belief that the ileostomy should be performed outside of the area of peritonitis. In the two successful cases this was accomplished by employing a left rectus incision.

Three of the four cases of obstruction developing months after appendectomies with drainage recovered by freeing of bands. One patient died of peritonitis from perforation of the obstructed bowel. Two patients recovered after freeing of bands originating in caseous mesenteric glands. No cases of obstruction from general adhesive tuberculous peritonitis were included.

In one patient, from whom an enormous mesenteric cyst had been removed, obstruction resulted from a combination of narrowing the lumen of the bowel and adhesion of the appendix to the site of excision. Enterostomy proved to be a life saving procedure in this case but had to be made so high in the small bowel that the problem of starvation became an acute one. This was met by collecting the food as it came from the enterostomy, reintroducing it through a catheter by the point of previous obstruction and early closure of the enterostomy. The convalescence was from then on uneventful and the end result satisfactory.

There was one case of strangulation of a loop of ileum through a defect in the mesentery. This patient recovered following resection and lateral anastomosis. One patient died of sepsis following an attempt at lysis of adhesions with enterostomy.

It is interesting to note that in these seventeen cases of obstruction from varied causes, enterostomy was performed six times with five successful outcomes. This is a great contrast to the failure with its employment in cases of intussusception. In this series it was only employed three times with one recovery. It has been resorted to by one of the writers and by other surgeons prior to this series without striking success.

Speculation as to the difference in results from the same operation in intussusception and obstruction from other causes brings up the question of what is the cause of death in intussusception. The operation for the reduction of intussusception is one which necessarily involves much handling of the bowel and it would seem might cause great shock, yet it is our belief, that these patients almost never die of shock. Dehydration may be and probably is a factor but not a very great one. These patients are usually of a type who should be able to stand considerable loss of fluid. They do not respond sufficiently well to replacement of fluid to lead one to the conclusion that this is the main factor. The theory that bacterial infection through the damaged intestinal wall is the main factor is not borne out by autopsy findings. Also the fact that the higher the obstruction the more rapidly fatal the outcome is against the bacterial theory, because bacteria are less numerous high in the intestinal canal than low.

The experimental work of Whipple and his associates [4 5] in this respect tallies well with the clinical facts. In animals in which obstruction had been produced they failed to demonstrate bacteria in the blood stream, liver, spleen or peritoneum. Whipple produced a toxin from a tied off loop which when injected into an animal proved rapidly fatal. He believes this toxic substance to be a proteose substance the exact nature of which is not determined. It is of particular interest to note that experimentally the animal with obstruction lives if the tied off loop of gut is drained. Eisberg's work[6] along this line is also of special interest and indicates that the toxic substance originates in the obstructed loop. All the experimental work suggests the advisability of drainage of the obstructed bowel to limit or dilute the toxic material causing the lethal outcome.

In none of the experimental work, however, is the question of temperature emphasized. Some of the animals died without fever. This is in striking contrast to the cases of obstruction es-

pecially intussusception in the human being. High temperature is one of the most striking phenomena of these cases. (Chart II.)

The febrile reaction in the fatal cases was extremely rapid, the thermometer registering one hundred and nine within twenty-four hours in one case. The high temperature and fatal outcome usually occur in the cases where operation has been too long delayed. To combat the toxicity causing this temperature is the problem at

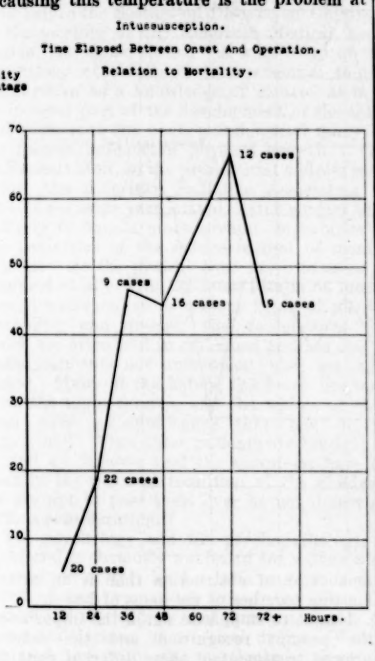

Chart I.

present. From experimental evidence it would seem that enterostomy should be an important part of our remedial procedures. From our own clinical experience this has proved to be true in cases of obstruction other than intussusception. In intussusception it has not so far had the success which would seem it should have. It is possible we may find that enterostomy performed higher up in the ileum may solve the problem.

The drainage of the thoracic duct as done experimentally by Costain[7] removing the toxins before they reach the blood stream is interesting and logical. The technical difficulty of this procedure in infants is sufficiently great to contraindicate its employment.

During the operation care in avoiding unnecessary trauma is important. No unessential procedures, such as appendectomy, should be performed. It is usually not necessary to suture slight tears in the serosa. At the end of operation salt solution with adrenalin has been given by the rectum with apparently beneficial results. Possibly salt solution alone might be as effective. Camphor in oil is usually used for stimulation whenever required. Colonic irrigations and the administration of rectal glucose are indicated in the after care.

Intravenous glucose and saline hypodermoclysis may be occasionally indicated but dehydration is not usually sufficient to indicate their use; rest is usually more important. Moderately early feeding should be resumed, though the amount should be small.

Early diagnosis and prompt surgical intervention remain the most important factors in reducing mortality[8]. Our results show that improvement is taking place in this community but there is still room for more.

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DISCUSSION: Dr. James S. Stone, Boston.

Dr. Ladd has put so well the opinions of all of us at The Children's Hospital that there is very little to add, and nothing to criticize.

I think we owe a tremendous amount to a monograph on intussusception published a good many years ago and lately republished by Clubbe of Sidney, Australia. It is interesting to note that in Clubbe's statistics of his first fifty cases there was a mortality of fifty percent, and of his second fifty cases his mortality was cut in halves, to twenty-five percent. His next series was cut in halves again, to about twelve and a half percent. The improvement in his statistics seems to be due practically entirely to the fact that he had educated the community of doctors in Sydney to a prompt recognition of the disease. Then later when the war came and many went away and those left were overworked, his mortality jumped right up again. There was a failure to treat the disease promptly. It is on the early recognition and early treatment of this disease that improvement must chiefly depend.

I want to emphasize one thing, that intussusception usually starts at or near the ileo-caecal valve. It may start at a Meckel's diverticulum or at a fibroma, or be the result of thickening in the Peyer's patches. The ileum goes through the ileo-caecal valve, and then passes up the ascending colon. Within the first few hours, or in other words at the time when the physician is likely to make his first call, the intussusception is commonly at the hepatic flexure under the liver where nothing can be felt. Clubbe em-

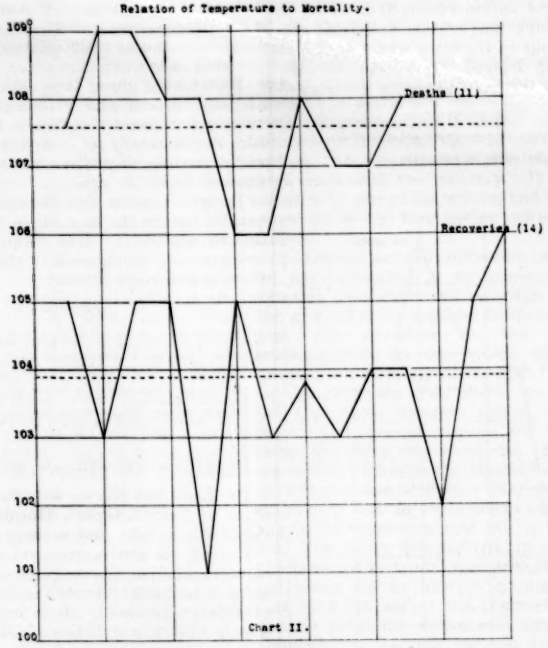
phasizes that it is very important to make an examination under ether if the history is characteristic and if no tumor can be felt. Cases should not be overlooked because the tumor is under the liver.

If the cases are recognized and treated promptly then the drainage of which Dr. Ladd has spoken is not necessary. The toxicity of the intestinal content comes with time and if the intussusception is reduced promptly the toxicity will not have developed. As Dr. Ladd has said, the results of draining the bowel have been disappointing so far in intussusception but we have not gone far enough as yet to reach a final judgment.

Various congenital anomalies and all sorts of inflammatory conditions may cause intestinal obstruction. One single band is a much more common cause of obstruction than is an extensive matting together of the loops of bowel.

I want to emphasize again the importance of the prompt recognition and the extremely prompt treatment of these different conditions, and I know no book that is more interesting than that little monograph of a hundred pages entitled "Intussusception, Its Diagnosis and Treatment" by Clubbe. It is something that everybody ought to have and read. It is a small book but a valuable one, and was written by a man who thoroughly knows his subject.

Intussusception.



	Cases	Recovered	Died	Percent Mortality	
C. H. Intussusception (1908 - 1915)	63	32	31	49. %	Ladd, W. E., Boston M. & S. J., 173:879, 1915.
" " (1915 - 1923)	88	60	28	31.8 %	Ladd, W. E. & Cutler, C. D. 1924.
" " "(Under 1 Yr.)	66	46	20	30.30%	"
" " (Reductions)	72	58	14	19.4 %	"
" 105 Cases Intestinal Obstruction	105	72	33	31.4 %	"

TABLE I.

MENTAL HYGIENE AND ITS RELATION TO PEDIATRICS

BY D. A. THOM, M. D., BOSTON, MASS.

DURING the past year, two eminent pediatricians have called attention to the importance of a better understanding of the mental life of the child and the relation between psychogenic factors and a variety of symptoms seen in children. Dr. Borden S. Veeder, in the Chairman's address read before the Section on Diseases of Children, at the meeting of the American Medical Association, last June, stated, "A knowledge of the psychology of childhood is as essential to the pediatrician as a knowledge of disease, as it is an integral part of the development of the child and without it one cannot understand many of the factors influencing physical growth." Dr. L. Emmett Holt, in the presidential address read before the American Pediatric Association in May of the same year, stated, "One subject that is likely to have a much greater importance in the pediatrics of the future is that of mental hygiene. At the present time the profession is occupied chiefly with the later results of faulty mental medicine, as in various types of mental instability and disease, the foundations for which are often laid in childhood and are due to maladjustments not understood and not corrected. Many of the habits, the fears, the tempers, the night terrors, and the whims of children have a significance that few of us appreciate. When these patients are brought to us and we discover that the symptoms have no basis in the physical condition of the children, we are apt to pass them over as not deserving serious consideration."

The psychology and the psychopathology of childhood is obviously too broad for a discussion in a paper of this kind. It is quite true that much of our present-day knowledge about the mental life of children is of purely academic interest and lacking in practical utility. Within the past ten years and particularly since the close of the War, a new and more intelligent approach has been made to the subject of mental health which has resulted in a clearer comprehension of many of the problems pertaining to child life. Practical demonstrations, through the schools, courts, and medical centers interested in children, point to the necessity of studying the individual as a composite unit. Important as it may be for the child's physical system to be operating in an efficient manner and to see that every care be given to teeth, tonsils, gastrointestinal tract, glands of internal secretion, to feeding and posture, and to regulation of hours for sleep and play, it is equally important to develop in this child an emotional life that is not twisted and warped by undesirable habits, personality defects, and conduct disorders that are apt to become woven into the fabric of the personality at a very early age.

I know of no better way of presenting the practical side of the subject of mental hygiene and its relation to the child than by outlining the organization and development of the mental clinics which were started in Boston about two years ago and to present a few typical cases illustrating their value.

The mental clinics have at all times been considered as just one branch of the medical work as developed by the Community Health Association. We never lose sight of the fact that a physical basis for undesirable habits and personality defects should always be considered first. The clinics are held at the community centers throughout the city, an effort being made to render this service in the most convenient manner. Operating the clinics in the community from which we are to draw the patients has many advantages. We have a more intimate knowledge of the facilities available in that particular section which are of service in solving the problem at hand. Each clinic acts as a center from which information regarding mental health can be disseminated. The importance of rendering the service with the minimum amount of inconvenience is a point worth stressing inasmuch as only a very small per cent of the patients seen at these units would ever have received any attention had the parents been required to make any great effort. Such problems as jealousy, cruelty, shyness, enuresis, refusal of food, etc., do not arouse the same concern in the minds of the parents as would a broken bone or a severe skin rash.

The family is always considered as the unit. This means that invariably the mother and one or more other members of the family will need to be treated and educated along certain lines before the patient's problem can be solved. The habit clinics are primarily therapeutic centers and not research laboratories and our function is to apply, in such cases where it seems advisable, to the general medical work a specialized knowledge regarding the mental life of the child.

The cases in which the mental clinics can be of most value divide themselves into four fairly well-defined groups so far as cardinal symptoms are concerned but invariably there is a considerable overlapping of one group with another.

Group A—Undesirable Habits Pertaining to the Organic Life of the Child.

EATING—
- Refusal of food
- Demands to be fed
- Finicky regarding food
- Perverted tastes, etc.

SLEEPING—
- Night terrors
- Walking in sleep
- General wakefulness

ELIMINATION—
Enuresis
Soiling
Refusal to permit elimination

SEX HABITS—
Masturbation
Unusual sex curiosity
Precocious sex tendencies, etc.

(Treatment invariably more injurious than the habit.)

Group B—Personality Defects
Shyness
Pugnacity
Jealousy
Day-dreaming
Selfishness
Fear
Temper tantrums
Domineering tendencies
Personality change following illness

Group C—Physical Manifestations
Peculiar mannerisms
Tics
Convulsions
Disorders of the special senses
Nail-biting, thumb-sucking, stammering, lisping

Group D—Conduct Disorders
Lying
Stealing
Disobedience
Destructiveness
Cruelty

These undesirable habits and personality defects, in order to be eradicated, must be replaced by some new interest. It is not sufficient simply to build up barriers which will thwart the desires and block the emotions acting as the driving force. This force must find a new outlet in a way which is compatible with the environment and satisfying to the child. He should be led and not pushed. All too frequently, it is the emotions that act as the stumbling-block rather than the intellect or the will. In order to dispense with undesirable habits and to form new ones, it is of paramount importance to find a motive for the rejection of the old habits and the acceptance of the new—that is, to substitute something in the place of the rejected habit. The idea of the new habit must be clear in the mind of the child. It must appear to the child as a thing capable of achievement and the advantages to be gained from its development must be presented in such a way that satisfaction will come with the striving as well as with the accomplishment.

There are no routine measures that can be applied to every case presenting personality defects or undesirable habits. Take, for example, the problem of temper tantrums in children. In John, temper tantrums were always associated with physical illness; in Mary, with extreme jealousy of her younger sister; while Henry utilized them knowingly and voluntarily to get his own way and he continued to use them so long as they worked out to his advantage. Another youngster reacts to intense anger with temper tantrums. In each case the symptoms were almost identical but the causative factors were quite different and the treatment in each case had to be brought about by a series of different adjustments.

Destructiveness may be due to cruelty or anger or perhaps to some vague, intangible mental conflict, deep-seated and of long standing, or possibly to some new and rather trivial environmental situation in which the emotional reaction has been excessive. These personality defects must be considered and treated pretty much the same as we would treat fever or headache—that is, make every effort to determine the cause and remove it whenever possible.

We have two distinct groups of cases with which to deal. The first group is the smaller but, by far, the most difficult to treat successfully—that is, the constitutionally neurotic child. When we are able to obtain the developmental history of this type of individual, we find that the child has always been hypersensitive and has shown exaggerated reactions to sensory stimuli, such as noise, light, heat and cold, wearing apparel, etc. Slight indiscretions in diet often produce marked gastro-intestinal upsets and skin reactions. Emotional upsets out of all proportion to what one might expect are stimulated by any strange or unusual situation, the emotion of fear usually being the most predominant. The infectious and toxic conditions of children are quite apt to produce severe reactions on the nervous system, extreme temperatures, tics, and convulsions. These children sleep poorly and suffer both physical and mental unrest. They find it difficult to get to sleep and also to sleep quietly once it is induced. Everything in their environment registers keenly. Out of the recesses of their troubled minds, they project strange, weird, and terrifying figures and if their imaginations cannot be contradicted by their special senses, they frequently pass through emotional disturbances which are, at times, overwhelming. As these children get older, we frequently find physical symptoms coming more to the foreground—neurotic vomiting, choreiform movements, stammering, convulsions, etc. Or it may be that the instability manifests itself in personality defects such as jealousy, shyness, pugnacity, cruelty, and feelings of inferiority. All these symptoms may occur during the first two or three years of life and much depends on the attitude of those with whom the child makes daily contact how important these mental reactions are to become in later life.

One of the most difficult tasks that a parent

has is in making certain necessary allowances for the constitutionally neurotic child, on the one hand, and guiding with intelligence, firmness, and fairness, on the other, the development of the child's life so that the innate neurotic tendencies will not be exaggerated and perseverated. One must also take into consideration the attitude of the child toward these symptoms because he, having been impressed with the fact that he is a bit different from the average child, frequently utilizes this difference as a means of gaining his own ends. In other words, one frequently sees the constitutionally neurotic child exploiting his symptoms with a well-defined motive in view.

The second group of cases represents those children manifesting neurotic tendencies who have previously shown none of the constitutional instability of the group just mentioned. These cases suddenly and without apparent reason develop many of the symptoms just described in the foregoing group. I say without apparent reason because invariably one finds, after a careful study, some environmental situation, some experience or series of experiences to which the child has been subjected and which accounts for the disorder in conduct that we call a neurosis. On the physical side, we find extreme cases of mal-nutrition, toxic conditions such as are brought about by influenza, meningitis, encephalitis, or trauma and which produce disturbances of the glands of internal secretion. On the mental side, we may have an excessive emotional strain such as earnest application to intellectual pursuits, being subjected to some experience where the emotional reaction has been intense such as terrifying situations in which fear is predominant, and a rather large and unrecognized group of cases where the emotional reaction has not been intense but has lasted over a long period of time.

The following cases are representative of the simplicity or complexity of the problems presented at the clinic.

M. C. is a very attractive little girl, ten years of age, whose medical history presents nothing worthy of note. (She is in grade 4A in school and getting on well, which may be taken as a measure of her intellectual capacity.)

She was brought to the clinic by her father who stated that she was "viciously destructive" and "wilfully stubborn." He gave me the following details concerning recent happenings.

All during the winter, she had persisted in going down cellar and turning on the cold water, permitting it to run into the steam boiler, thus cooling off the house. For this she was scolded, threatened, spanked severely and finally her bare hands were placed on the hot furnace, blistering them severely and necessitating bandages for several days. Fifteen minutes after their removal, the act was repeated.

Four days previous to her visiting my office, for some unknown reason she took a pin and scratched the piano. This episode was followed the next day by mutilating the top of the dining room table with the cover of a tin can. For these offences, the father scarified the palm of her right hand and arm with a pin, leaving ugly looking wounds which were much in evidence when I saw the child.

Recently her father missed several graphophone records and upon appealing to the patient, she admitted taking them to school, but upon her father's request did not return them. He went to the school with the patient and interviewed the teacher and principal, to whom, the patient stated, she had given the records, only to have her admit later that she had lied. She was punished severely, but maintained a sullen silence until the next day when she told the housekeeper that she had put the records down through the cracks in the veranda. A carpenter was secured and several boards were removed, but no records were to be found. A few days later, of her own accord, the patient produced the records which had been hidden away in her room.

She writes on the wallpaper, hammers the walls, and destroys furniture.

She is the oldest of five children whose mother died three years ago. Her father states, "I have had twenty housekeepers since then." The one in charge now is sixty-three years old; she is kindly and affectionate toward the patient and the child is fond of her. The father is a stern, reserved, quick-tempered man who is trying hard to keep his family together and, in spite of his apparent brutality, wants to do what is right.

At school, the child is considered kindly and affectionate, bright, well-behaved, and truthful. At home, she is untruthful, disobedient, destructive, selfish, and jealous of material things, not affectionate, stubborn and resentful. Her father states, "She is willing to undergo any pain to exasperate me."

In the office, we find her to be a happy, cheerful little girl who admits frankly her jealousy of her younger sister. She gleefully tells about school-day experiences but suddenly becomes sad and tearful at the mention of her mother. She assumes the responsibility for "all the rest of the kids," as she calls them. Is interested in schoolmates. Wants pretty clothes. Likes her teachers, the housekeeper, and her father. Shows no resentment for the severe punishment she has received. Offers no excuses or explanations for her misconduct.

She seems to be very friendly and accessible. One feels that there is a sympathetic relationship established which will do much to get things going right. The father is given to understand that punishment is useless, which fact he has appreciated for some time. He is asked to get on

a more companionable basis with the children and a few days later he demonstrated his good intentions by bringing in three of the children to the office, enroute for the movies. The report of the father at this second visit was quite encouraging. Patient had been getting along fine for a week, none of the destructive tendencies being in evidence. She seemed happy and cheerful and talked more freely; she was much overjoyed at the prospect of going to the movies.

After the picture show, the patient returned home. Everything seemed to be progressing well when suddenly, for no apparent reason, the child gathered up several phonograph records and destroyed them. There seemed to be no particular emotion attached to this episode, apparently being the result of an impulsive idea. She was not punished on this occasion and everything went along smoothly for forty-eight hours, her father still hopeful that another week might pass by without further manifestation of her destructive tendencies. The second evening he brought home a new pair of white shoes for the patient, a present for which she had shown a strong desire for some time. She was happy over the gift and seemed very appreciative, but within an hour after her father's return, she cut the upholstering on one of their best living-room chairs with a pair of scissors. This information was given me by the father over the telephone. He admitted that he had reached the limit of his patience and some plan must be made whereby the child would be taken from the home.

Arrangements were made with the New England Home for Little Wanderers in Boston to take this child for an indefinite period, and although the father had demanded that such a plan be made, he let the matter drop at that point.

Comment. The case was not under my personal observation sufficiently long to enable me to formulate any definite ideas as to the underlying mechanism for the cause of her destructiveness. There are, however, several pertinent factors in the history that give one an inkling of the line of treatment that must be followed. The first and most important is the child's devotion to the mother, her inability to assimilate into her own life the emotional experiences caused by the mother's death, and her bitterness and resentment on being deprived of her mother.

In a superficial examination of the facts presented, we find that all the destructive tendencies of this patient are directed toward the house in which she lives and the furniture contained therein. At school, when visiting, or under any circumstances or conditions not found in her own home, she never manifests any of these destructive tendencies. It seems that it is the association with her own home that brings out all of her vicious tendencies. One also finds from the history that, for many years prior to the death of the mother, the sole interest of both parents was to save enough money to build a house that would be more or less of a show place in the community in which they lived, a small unattractive village in the suburbs of Boston. Both parents worked without daily recreation or vacations, and even went without the necessities of life until the death of the mother. Frequently they denied themselves the essentials of life in order that another dollar might be put away. It was, however, only after the death of the mother that the father finally erected this $16,000 house which stands out as a monument of his ambition and thrift. But somehow one cannot help but feel that, to the patient, consciously or perhaps unconsciously, it is but a memorial to the toil and sacrifice of the mother to whom she always showed a very strong attachment. This may be considered merely speculative, considering the opportunity I had to observe this particular case, but it is not unreasonable to expect to find an explanation for these apparently voluntary vicious acts of destructiveness in some deep-seated mental conflict with which the child is struggling blindly.

A. C., a little girl, aged 4½ years, was referred to the clinic because of temper tantrums followed by peculiar spells characterized by violent paroxysms of coughing and apparent loss of consciousness and rigidity of the muscular system, but with no convulsive movements.

The patient is a very attractive youngster, extremely affectionate, generous, unselfish, but inclined to be rough and pugnacious towards other children with whom she plays. She has numerous ordinary fears such as fear of animals and of the dark but no definite phobias. She is very active and the mother states that she is never quiet a minute.

There is nothing of importance in the medical history prior to April, 1923, when she had a severe attack of whooping cough which lasted until September of the same year. From the mother we learn that it is only during and since this last illness that the patient has been difficult to manage. Since that time the temper tantrums and so-called spells have occurred several times a day and they are invariably associated with situations where the child is unable to have her own way. They occasionally occur for no apparent reason at all. She has a "spell" invariably when she is scolded and always when she is punished and when teased by other children or when interfered with when teasing them. In fact, the child, during the six months prior to coming to the clinic, had been meeting every difficult situation in this way.

She was referred to the Children's Hospital in order that any physical basis for the paroxysmal coughing might be determined, but the physical examination proved quite negative. The problem was then considered as an undesirable habit which the child was using for two reasons—to

gain, first, her own ends and, secondly, the attention of the mother for whom the child had an unusually strong attachment. The mother's concern regarding the seriousness of the spells was minimized and she was educated to change her attitude toward the child while the latter was having a spell. Every effort was made to make the child understand the undesirability of such tantrums and during the next two weeks, she had only one spell. At the end of six weeks, the mother reported that the child was perfectly all right and there is now no problem regarding discipline.

Another case of similar type is that of a lad, nine years of age, who was brought to the Out-Patient Clinic at the Psychopathic Hospital. His medical history was not important, until four months previous to his first visit when he had been knocked down by an automobile and taken to a general hospital where he was treated for a period of two weeks. His condition at that time was considered serious and he had a rather long convalescent period at home during which time the entire routine of the household began to revolve around him. The mother was extremely solicitous. Every whim of the child regarding his food was gratified. The other children were told that he must not be annoyed and all the toys were his to accept or reject as he wished.

For the first time in life, the lad found himself in the limelight. The situation was naturally not unpleasant and many were the motives presented for the apparent continuance of the effects of the accident. The mother stated, "He has become very surly, always looks ugly, seldom smiles, appears unhappy and discontented, and acts as if he wanted to be left alone. With the other boys he has become selfish and domineering, a poor sport, and they have started to call him 'empty-head.'"

After a careful physical and neurological examination which was entirely negative, it was decided to change the entire regime of the household. Notwithstanding the fact that the mother had seven children to look after and was an uneducated Swedish woman, she comprehended exactly the coöperation necessary on her part. It was a matter of only a few weeks before she reported that the patient was happy and contented, played with the other boys, was getting on well in school, and had lost all his sullenness. There was no further evidence whatever of the personality change which was apparent when he first came under observation.

The two cases just cited are good examples of how certain mental reactions in children may be exaggerated and perseverated by the oversolicitous attitude of the parents.

D. H., a little girl of 6, was brought to the clinic by her father and school nurse because of voluntary defecation and other associated undesirable habits. The patient's birth and developmental history was negative.

She is the youngest of three children, the other two being in excellent health but manifesting unusual neurotic tendencies. The older brother, aged 12, has passed through experiences almost identical with those of the patient. Over a long period of time he soiled himself, often at times when requested to do something contrary to his wishes. He was sexually precocious and imparted much distorted sex information to the patient and admitted sexual relations with her on numerous occasions. He hated school and frequently utilized the soiling method to avoid it. He had stolen small sums of money. The sister, Margaret, aged 9, was a persistent masturbator.

When the patient was 9 months old, her mother, a fairly intelligent and respectable woman, died. The mother was the illegitimate child of a woman who is now looked upon as a notorious character, irresponsible and abusive, and who, interestingly enough, is reported to have had the habit of soiling her clothing for over a long period of years. After the death of the patient's mother, the children were moved about from one home to another until in July, 1922, the father secured a housekeeper, a woman who has separated from her husband and has with her her two children, a girl and boy, aged 13 and 10 respectively. The household now consists of the father, the housekeeper and the five children of the two families.

At school, the patient is in the first grade and is doing excellent work. She is said by her teacher to be the brightest child in the class.

The following is the father's account of the patient's present difficulty:

In spite of intensive training (which one may rightly question) the child has never established proper toilet habits. She has rarely missed a day when she has not soiled her clothing and sometimes this occurs two or three times a day. It is quite obvious to the family that it often occurs as a response to some unpleasant situation or as a means of gaining revenge. Not infrequently, she will soil her clothing and then contaminate a fresh supply of laundry, taking piece by piece and smearing it. It appears that she prefers to have her bed and underwear smeared rather than clean. She has marked perversions of taste and will eat horse and hen manure without being in the last abashed. She even comes to the table with her hands smeared with feces and, although complaining of the odor, will put her hands in her mouth as a means of cleansing them. Food in the house has to be carefully watched to prevent her from smearing it.

The father further states that he would be inclined to feel that these habits are beyond her control were it not for the fact that she has evidenced brief periods when the habit was not present as shown when, on a visit to her grand-

mother's, it was necessary for her to leave her bed in the middle of the night and go to an outside toilet. On various occasions she has stated that she could get over the habit if she felt so inclined but that she does it for spite.

She has boasted of her sex activities and sex knowledge and on one occasion was found fondling one of the male employees in a very suggestive fashion.

She is extremely destructive and gets no enjoyment whatever out of the usual toys intended for a girl of her age. Last summer she uprooted her father's garden and she has removed all the ivory keys from the piano.

Notwithstanding the fact that she is an outcast from her family circle, she does not appear to be at all humiliated but is quite indifferent to the approbation or criticisms of those with whom she is living. Disciplinary measures avail little or nothing. The only favorable comment the father makes regarding the patient is that she is a very lovable child.

From the ability that the child has shown to get along in school and on account of her psychological examination which gives her a mental age almost two years beyond her chronological age and an intelligence quotient of 125, one cannot account for this conduct on the basis of mental defect.

In closing what has necessarily been a very sketchy outline of the relation of mental hygiene to pediatrics, I feel that, although many of the problems relating to the mental life of the child are perhaps so vague and intangible and their treatment so time-consuming as to make it impractical for the pediatrician to include this group in his practise for treatment, a very large per cent of all the problems relating to the child's mental life are not of this complex nature and they are being treated intelligently and skilfully not only by pediatricians but by intelligent parents and teachers. I do feel, however, that the mental hygienist already has a certain amount of information regarding the mental life of the child which should be included in the curriculum of our medical schools under the section of pediatrics.

DISCUSSION: Dr. Bronson Crothers, Cambridge. The thing that I feel after seeing psychiatrists in the work for several years is that as a class they underestimate themselves. I think that they have a tendency to attribute to technique, results which really come from their extraordinary intelligence and devotion to the cases and from the conception of education which they are able to bring about. Dr. Ladd has shown us that you must treat intussusception and emergencies of that sort promptly, but these cases must be given more time. When a parent comes into a doctor's office with the ordinary problems of childhood the tendency is for her to feel that the doctor ought to treat the child as he does other cases and be able to give advice before they leave the office, so the doctor is not in a position to spend a great deal of time to find out what the difficulty is. A great many of these disorders have very trifling symptoms, but doctors have to settle down and work with them just the same. Then too it is quite easy to develop, in reading certain extreme psychiatric literature, a feeling that a great deal must be poppy-cock because it does not fit in with the experience that the physician has. Dr. Thom realizes that almost never can a doctor straighten out a child in one visit. The only possible chance that he has to get results is to find out more about it than the mother can tell at the first visit. Even the mother can't tell everything that should be known. If she knew it all she would not have to come to see the physician.

My impression is that the chief bar to successful mental hygiene or successful treatment of these conduct disorders is not necessarily lack of technical facilities or psychiatric technique. The conduct problems involve the whole family and many doctors don't want to take the chance of antagonizing the family. Some of these problems cannot possibly be settled without the intervention of a social worker, and a great many people feel that a social worker sent into the home is a nuisance, but everyone must be made to realize that he should welcome having somebody come in and find out more about the child, its surroundings, the home, the servants, etc. I think it is conceivable that a method of approach could be worked out for many of these problems without any elaborate psychiatric technique. To my mind the great contribution of psychiatry to pediatrics is the recognition that the doctor is called in to direct a process which is infinitely more complicated than most of the physical diseases.

The treatment of these cases is necessarily prolonged. I think it is quite significant that in twenty minutes Dr. Thom was able to outline the problems, whereas even with the extra time allowed he was able to merely suggest the means by which he reached extremely satisfactory results. These cases tax, more than anything else, the patience, and if we think we only need to call into play the various psychiatric and psychological technical methods we won't get very far.

SECOND DISCUSSION: Dr. Daniel J. Fennelly, Fall River. I became interested in this particular part of mental hygiene through the Juvenile Court in my city of Fall River. About nine years ago Judge Hanify asked me to help out on some of their mental problems, and, having no way of getting what might be considered really successful results without court authority in these cases, I was designated as a voluntary probation officer connected with the Court. So for nine years I have been rather intimately connected with this phase of the work,

not probably as well as it has been handled by Dr. Thom, and the various angles that have been treated during this period have evolved in my mind something that has not been entirely covered by his paper.

We hear a lot about taxation and the amount of money that is being expended by government, so I would bring this home to the men here from that point of view: do we realize the importance of the fact that 20 per cent of all the money that is collected in Massachusetts goes for the care of the mentally sick, and that almost three-quarters of the rest of the money is devoted to the education of our various children in the different communities! If you realize also that the cases that are in mental institutions are of those who either have been committed or are children of the feeble-minded class, you may also consider that these are the cases which you and I and the majority of men view as hopeless ones.

Here is a side that has been covered this morning which concerns the children who are not feeble-minded and who are not really mentally unbalanced and can be helped. It would seem to me as though the majority of the pediatricians and general practitioners of the State should be interested in this thing from the money point of view. If we are spending all this money on the care of people in institutions, why would it not be better to get down to the root of things and prevent some of this? It is in this way that Dr. Thom and others in this great field of usefulness are doing a very valuable work.

That there is a lack of interest on the part of medical men in general practice was called to my mind during my attendance at a hearing in Boston last month, when a branch legislative committee was asked for an appropriation for the extension of mental hygiene. I believe that there were just five medical men present at the meeting. In the section of Hospital Administration yesterday a paper was read which stated that one out of every 25 persons in the Commonwealth of Massachusetts had either been or was now an inmate of a State hospital or a sub-division of a State hospital. We ought to realize how tremendously important this problem is in adults. These cases are all potential cases for institutions if they are not taken care of. If from a mental hygiene point of view we can educate the doctor to take care of these subjects in childhood, we are going to save the State a lot of money some day.

THE DIAGNOSIS OF ENDOCRINE DISORDERS

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THE present science of endocrinology, if it may be so designated, is in large measure a product of the 20th century. Prior to 1900 certain well defined clinical entities, designated largely by the names of those who had first described them, were recognized and their endocrine origin clearly established. In the majority of instances, however, these syndromes represented the end results of an endocrine pathology unrecognized in its earlier stages. In the last quarter century, however, in part as the result of skilful and elaborate animal experimentation and in part as the fruit of keen and extensive clinical study, a much clearer concept of the diverse functions of the endocrine system has obtained. Experimental pharmacology and pathology, physics and chemistry have each added to the sum total so that today one may feel, with due conservatism, that a beginning has been made in the solution of the endocrine problem.

The present paper deals with certain of the results obtained in an extensive clinical and laboratory study designed to establish, if possible, an objective method of determining aberrant endocrine function. No attempt is made at this time to present a review of the voluminous and, in part, uncritical literature of clinical endocrinology. The several admirable compilations which have recently appeared make this unnecessary even did space permit.

While general endocrine disturbance is usually recognized, the determination of the individual focus of disturbed function offers far greater difficulty. Starting from the common point of the clinical picture, individual observers arrive at widely divergent conclusions in regard to the etiology of the condition. And as diagnosis is an absolute prerequisite for the replacement therapy required in these cases, the question of etiology becomes one of basic and primary importance.

In the field of so-called vital function testing a method of great possibilities is presented. True, many of these tests have been discredited and discarded because experience has shown them to be non-specific. When one considers the multitude of sensitive mutually dependant equilibria which determine the level of functional performance of the human body, it is obvious that this objection is not valid. A wholly specific test is hardly to be hoped for. Even the highly differentiated tests, in the field of immunology, sometimes fail of a complete specificity. The Wassermann is a case in point. Approaching the problem from the other point of view, this non-specificity offers the largest factor of value. The selection of not one, but of many such tests, and the determination of normal response to each of them, establishes a series of definite criteria. The next step is the selection of subjects in whom exists so well marked a pathology localized in a single focus as to leave the true diagnosis in no doubt. In this group come the surgical castrates and hypothyroid cases, advanced cases of exophthalmic goiter and acromegalia. Application of the whole series of tests to each of these

experimental subjects indicates both the sense and magnitude of departures from the several base lines, induced by a modified function of the single focus.

The third stadium rests in the study of the individual case, the classification on the basis of the laboratory findings only after all non-endocrine pathology has been carefully ruled out, the exhibition of the single indicated glandular extract with continued clinical study and repeated laboratory tests. The writer cannot stress too strongly the necessity of eliminating non-endocrine conditions as a preliminary to the establishment of an endocrine diagnosis. Blood uric acid is significant only in the proven absence of nephritis and gout, eosinophilia when no protein sensitivity exists, lowered sugar tolerance in the absence of diabetes, to name but few of the many. For this and other reasons, no single test is independently significant but derives its meaning only in relation to its fellows. Finally, clinical and laboratory findings supplement and complement each other. Both are essential.

Certain other points should also be touched upon. The present experimental studies have been confined entirely to man. Both the wide divergence of the human race from the other mammalia in many of its metabolic processes as well as the possibly misleading response of severely traumatized and moribund experimental animals conditioned this selection. The possibility of the direct translation of the results obtained, in the writer's opinion, more than compensates for the obvious limitations.

Further, in the selection of methods accuracy has been the sole criterion. Time consumption has played no part in the decision. The methods for blood and urine analyses of Folin and his associates are used almost entirely. Basal Metabolism is determined with the Benedict-Collins Unit. S. R. Benedict's methods are used in the Tolerance Test. Tests have been retained or discarded as results indicated their significance or independability. For example, the determinations of so-called blood sugar curves was discontinued when their lack of diagnostic meaning had been adequately proven.*

Other observations have developed a diagnostic significance which initially could not have been predicated for them.

The plan of study may be briefly outlined as follows:

Cases of suspected endocrine pathology are referred to the clinic by the physician; a brief preliminary survey, which might well include a few simple laboratory tests as the Basal Metabolism, Blood Morphology, etc., gives in a few cases a positive diagnosis (thyroid cases), in a much larger number reasonably clear indications that the case is not endocrine and in the residual group leaves the question undecided.* This latter group is then referred for the abridged study, a brief report of the details of which has recently been made.†

The allocation of a group of cases receiving this form of study is given in Table I.

TABLE I

SERIES S, SUMMARY

Diagnosis		No.	%	Group %
Not Endocrine		85	29.6	29.6
Pituitary	—	33	11.5	
"	±	26	9.1	22.0
"	+	4	1.4	
Thyroid	—	17	5.9	
"	±	9	3.1	10.8
"	+	5	1.8	
Gonad	—	44	15.3	15.3
Adrenal	—	3	1.0	1.0
Pluriglandular	—	4	1.4	
"	±	5	1.8	3.2
Unclassified		52	18.1	18.1
		287	100.0	100.0

The terms "hypo" and "hyper" are used more or less arbitrarily. When the whole level of functional activity seems to be lowered, the first is used; when the converse obtains, the second. Dysfunction is applied to those cases lacking the clearcut uniform definition of the first two. As will be seen, approximately 50% of the cases demonstrate an endocrine pathology, 30% are primarily not endocrine and nearly one-fifth of the cases remain unclassified. Where possible this latter moiety is referred for the Long Form study. This implies a 7-day stay in the hospital under controlled conditions of diet and observation. A large number of tests are carried out in duplicate, all indicated consultations are secured and as thorough a study made as is possible. The standard regime can best be presented in a condensed tabular form.

TABLE II

SCHEDULE

Routine	Special
First Day	
Collect urine I	
Blood Morphology	
History	
Physical Examination	
Second Day	
Phenol-Sulphone-Phthalein	Liver function with P-T-P
Salol	Protein Sensitivity
Adrenalin Mydriasis	
Alveolar Carbon Dioxide	
Special tests	

*Reported before the Am. Chem. Soc. in April, 1923.

*By an unequalled coöperation on the part of the writer's colleagues, it has been possible to allocate a certain proportion of the cases to the proper diagnostic group without further delay.

†See Rowe, B. M. and S. J., 190, No. 8, 1924.

Third Day

Collect Urine II	Respiratory Quotient
Basal Metabolism	Stool Examination
Vital Capacity	X-Ray
	Neuro Otological
	Audiogram
	Pelvic Examination
	Chest Examination
	Laryngological Exam.
	Measurements

Fourth Day

Basal Metabolism	Ophthalmological Exam
Vital Capacity	Drug Tests I
Repeat 2nd Day	McLean Index
	Cystoscopic Examination

Fifth Day

Blood Morphology	Heart Tracing
Blood Chemistry	Lumbar Puncture
Wassermann	Drug Tests II
Galactose Tolerance I	
Provocative Urea test	

Sixth Day

Galactose Tolerance II	Stool Examination
	Adrenalin Glycosuria
	Drug Tests III
	Psychometric Examination

Seventh Day

Galactose Tolerance III
Discharge patient in P.M. or start Mosenthal diet
Phenol-Sulphone-Phthalein

Eighth Day

Complete Mosenthal test, or
Galactose Tolerance IV, or
Duodenal function test

Orders for the work are based upon an expanded form of this schedule, with the deletion of non-indicated or addition of supplementary tests as the individual case requires. The diagnostic allocation of a second group of cases, given this more extended study, is collated in Table III.

TABLE III

SERIES B, SUMMARY

Diagnosis		No.	%	Group %
Not Endocrine		26	16.3	16.3
Pituitary	—	17	10.6	
"	±	39	24.3	36.8
"	+	3	1.9	
Thyroid	—	11	6.9	
"	±	9	5.6	12.5
Gonad	—	33	20.6	20.6
Adrenal	—	5	3.1	3.1
Pluriglandular	—	4	2.5	
"	±	6	3.8	6.3
Unclassified		7	4.4	4.4
		160	100.0	100.0

As is to be expected, the unclassified fraction assumes minor proportions. Also it is not uninteresting that in a group of cases which have been most carefully selected, one-sixth are shown to be non-endocrine although definitely suggestive from the clinical standpoint.

A correlation of the two series is informative. Too great weight, however, should not be attributed to the absolute percentages obtained. With so short a series (less than 500 cases), certain extraneous factors, which cannot be evaluated, play a part in determining numbers. With the established endocrine cases—50% of the combined series,—the partition is suggestive if lacking a final authority. The results are collated in Table IV.

TABLE IV

CORRELATION SERIES B AND S

Diagnosis	Short Form	Long Form	Summary
Not Endocrine	29.6	16.3	24.9
Pituitary	22.0	36.8	27.3
Thyroid	10.8	12.5	11.4
Gonad	15.3	20.6	17.2
Adrenal	1.0	3.1	1.8
Pluriglandular	3.2	6.3	4.3
Unclassified	18.1	4.4	13.2

In view of the extensive literature on the pluriglandular syndromes, the figures given above are most suggestive. A further analysis of this group offers even more striking evidence of the apparent rarity of these conditions.

TABLE V

ANALYSIS OF PLURIGLANDULAR CASES

	Short Form	Long Form	Total
Surgical	4	5	9
Doubtful	4	1	5
Functional or Organic	1	4	5

Practically half of these cases are pluriglandular only through surgical intervention and of the residuum but 5, or 1% of the whole, fall in this class with certainty.

With the large amount of data involved in the evaluation of each case, a statistical method of study becomes imperative. As, however, each diagnosis rests upon the inter-relationship of the many individual observations, such a general compilation in some measure defeats its own end and tends to obscure the real findings. To illustrate: A nephritis superimposed upon a gonad failure, may cause a rise in blood uric acid which is not diagnostic of the latter condition and thus makes it apparently comparable to a pituitary condition when the increase is really significant.

Further, in certain types of dysfunction, the blood is frequently either definitely lymphoid or leucoid in character. In any summary, these opposing conditions tend to nullify each other and mask the really significant divergencies. On the other hand, due allowance for these several factors entails so great a complexity in the presentation as to nullify the value. With a recognition of the limitations of this method of comparison, only certain of the more significant data can be presented in tabular form. To avoid further complications only the Long Form Series (B) is used.

bly from the norm entails uncertain extrapolation and leads frequently to absurd results. The chest standards are particularly dubious as increase in weight usually conditions a like change in chest circumference without increasing lung capacity. Using the chest standard, with abnormal fat distribution, one may readily obtain the anomaly of a short person weighing 250 pounds, apparently 10% under weight. The influence on calculated lung capacity is only less dubious and operates in the opposite sense. The West standards based on Standing Height and Area give more concordant results, although the Area

TABLE VI

PHYSICAL MEASUREMENTS

		Not End.	Pit.	Thy.	Gon.	Adr.	P. G.
Sex	Male	18	30	8	2	2	1
	Female	8	29	12	31	3	9
Age (years)	High	72	63	61	63	62	63
	Low	10	10	8	18	15	22
	Aver.	40	30	41	33	36	43
Height (cm.)	High	182	188	181	174	174	188
	Low	140.5	130	131.5	153	158	159.5
Weight (kg.)	High	92.7	124.6	114.8	110.0	89.0	106.0
	Low	28.1	21.6	37.0	38.5	48.2	55.0
Lung Vol. (cc.)	High	5500	5040	4520	3800	4100	3560
	Low	1480	1730	1100	930	2200	1720
B. P. Sys. (mm.)	High	238	200	146	184	98*	170
	Low	90	92	94	88	90	102
	Aver.	123	114	114	115	95	123
Dias. (mm.)	High	154	118	100	94	60*	90
	Low	45	50	52	50	14	64
	Aver.	70	72	77	73	43	73

*Omit case of hypertension.

PHYSICAL MEASUREMENTS

The physical measurements are presented as indication of the wide variety of experimental subjects. High and low values are given and when averages are significant, they are included.

The several blood pressure findings are perhaps the most informative of the data presented. Hypotension seems to be a common condition in endocrine states.

VITAL CAPACITY

Dreyer's interesting monograph* and the subsequent generalization of West† offer a basis for certain comparisons. In the use of Dreyer's tabulations only the standards referred to trunk height are used. The data were compiled apparently from average people and are straitly applicable to them only. The use of the other standards with individuals departing appreciavalue is influenced at times unduly by the weight of the subject. They never lead however to the extreme conditions found with the Dreyer measurements. The correlation between Dreyer's Trunk Height and the average of the West's length and area standards is usually excellent. Figures as given for Vital Capacity are averages of West and Dreyer final results. The data are given in Table VII.

*The Assessment of Physical Fitness, Hoeber, 1921.

†Arch. Int. Med., 25-306, 1920.

TABLE VII

VITAL CAPACITY

		Not End.	Pit.	Thy.	Gon.
Weight	High	+55	+88	+70	+107
	Low	−30	−30	−29	−37
	Aver.	−1	+7	+15	+4
Chest	High	+23	+49	+41	+66
	Low	−13	−24	−15	−16
	Aver.	+1	+1	+7	±0
Vit. Cap.	High	+12	+18	+11	+10
	Low	−62	−39	−70	−57
	Aver.	−23	−16	−28	−24

BASAL METABOLISM

This test is one of the most valuable in the series as it is significant both in sense and amount. Broadly speaking, lowered function is reflected directly in this measurement while overactivity produces a similar increase. The magnitude of the change, however, differentiates the thyroid from the other members of the group, conditioning as it does the most significant divergences from the base line. In measuring Basal Metabolism by the method adopted, all errors save the inexcusable one of exhaustion of the soda lime, operate to raise the observed rate. Values are to be regarded as maxima and only the most observant care and skilled technique suffice to exclude grave error. The so-called open method is equally open to the intrusion of vitiating error. For work of this character, the writer prefers the method adopted.

A second complication exists in the choice of standards. The writer's general practice is to use both the Harris-Benedict and du Bois and record the average of the two. In the majority of cases with adults, the correlation is good. With children and individuals of unusual contour where wide discrepancies exist the Harris-Benedict values are preferred. The data are given in the following table. Averages for Pituitary and Thyroid dysfunction are divided into positive and negative groups.

TABLE VIII

BASAL METABOLISM

	Basal Metabolism Deviation		
	High	Low	Average
Not Endocrine	+27*	—14	—1
Pituitary —	—4	—20	—12
" ±	+33	—25	+15 and —9
" +	+23	+15	+20
Thyroid —	—22	—50	—36
" ±	+25	—25	+25 and —15
Gonad —	—3	—25	—11
Adrenal —	+4	—10	—5

*Lymphatic Leukaemia.

URINE EXAMINATION

Of the many tests made with the urine, only a few permit of significant transcription in this tabular form. These are grouped in Table IX.

TABLE IX

URINE ANALYSIS

	Not End.	Pit.	Thy.	Gon.	Adr.
Urine Vol. cc.	1190	1150	910	1080	1270
Sugar % positive	27%	24%	10%	21%	40%
Salol (min.)	95	74	98	100	124
Urobilinogen % +	0	34	5	0	0
P-S-P % in 2 hrs.	57	57	51	59	48

The significant features may be summarized as follows:

1. An apparent lessened elimination in Thyroid cases, the Gonad group falling in the mean position. The predominance of women in this latter group offers a patent explanation.

2. One-third of the Pituitary cases give a positive response to the Urobilinogen Test of Ehrlich with dimethyl amino benzaldehyde. This cannot be interpreted as indicating a disturbed liver function but rather emphasizes the non-specificity of the test. The substance giving the positive reaction is at present unknown.

3. Only the Pituitary cases show an apparent normal gastric motility. The divergences are too slight and the test itself too crude to allow the observation to assume much significance.

4. The appreciable percentage of Melliturias in this group of non-diabetic cases supplements and enforces the observations of earlier writers. This should have some bearing on the life insurance problem.

5. The relative uniformity of the Phthalein test is broken only by the Thyroid and the few Adrenal cases. There are not enough to warrant any conclusions at this time. Comment has been made elsewhere (l. c.) on the existence of a pseudo nephritis in hypothyroidism which disappears under thyroid therapy.

An interesting point which this work has brought to light is the incidence of a high residual nitrogen fraction in the urine of endocrine cases. The composition of this moiety is at present unknown and investigation is now in progress to ascertain if it be an increase in normal constituents usually present in small amounts or the appearance of abnormal nitrogenous material. The data are given in Table X.

TABLE X

RESIDUAL NITROGEN*

Not Endocrine	12%
Endocrine	
Pituitary	50%
Thyroid	45%
Gonad	45%
Misc.	71%
Total	50%

*9% is taken as the upper normal limit.

BLOOD CHEMISTRY

Another possibly significant point which may assume diagnostic importance is found in the high blood uric acid values in the Pituitary cases. The few Pluriglandular cases are added to this group for this comparison as most of them have a pituitary element.

TABLE XI

URIC ACID TABLE*

Class	Gross	Net
Not Endocrine	50%	28%
Pituitary / Pluriglandular	63%	74%
Thyroid / Gonad / Unclassified	37%	26%

*Adrenal omitted as number too small to affect totals substantially and 3 of 5 doubtful as to classification.
4.0 mgm. is taken as upper normal limit.

In the above tabulation, the net values are obtained by the deletion of all cases showing a nephritic element. Many of the non endocrine cases had a nephritic complication which masked the true relationship.

The blood sugars are also interesting as the non-endocrine cases show a level of 102 mgm. while the endocrine averages are all below 90. These are the cases which show a very appreciable percentage of melliturias.

BLOOD MORPHOLOGY

Two points here are particularly significant. First. Many endocrine cases show a definitely lymphoid blood, there being both a relative and absolute lymphocytosis with substantially normal leucocyte count. (The average of the latter is about 8,100.) This is shown in Table XII.

TABLE XII

LYMPHOCYTES*

Not Endocrine	35%
Endocrine	
Pituitary	60%
Thyroid	68%
Gonad	25%
Misc.	76%
	56%

*33% is taken as upper limit of normalcy.

Only the Gonad cases fail to show the prevailing endocrine tendency. It may be added that there is a large degree of correlation between a high lymphocyte count and an increased residual nitrogen fraction in the urine.

The Eosinophile content duplicates the general observations of the Lymphoid elements. As never less than 500 cells, and usually 1000, are counted in a differential examination, the magnitudes of the less common elements have a real meaning. It is curious that the Gonad cases again group with the non-endocrines. No explanation can be offered at the present time.

TABLE XIII

EOSINOPHILIA*

Not Endocrine	27%
Endocrine	
Pituitary	49%
Thyroid	41%
Gonad	25%
Misc.	50%

*Skin diseases and protein sensitized cases omitted.
3% is taken as the division line.

GALACTOSE TOLERANCE

The threshold of tolerance for galactose offers a most important differential diagnostic test. The sugar is administered to the fasting patient at 7 A. M. and one two-hourly urine collection is made before, and several after, taking the test meal. Depending on the results of the first test, the dose is increased or diminished until the level is established at which melliturla first appears. The preliminary study on normals is now in press and a subsequent communication will give full details of the method. The threshold for the normal male and female differs by 10 grams so that results as reported must make allowance for the sex difference. The combined data for this series are given in the following table.

TABLE XIV

GALACTOSE TOLERANCE

		Male			Female		
		High	Low	Aver.	High	Low	Aver.
Not Endocrine				30	—	—	40
Pituitary	—	70	30	50	>100	40	64
"	±	30	5	16	80	5	63 and 17
"	+	20	5	12	—	—	—
Thyroid	—	60	30	43	60	30	43
"	±	30	20	24	50	30	38
Gonad	—	30	30	30	30	10	21
Adrenal	—	30	30	30	50	5	28
Pluriglandular	—	—	—	—	60	5	—

The results as given show certain salient differences. In the first place, the normal level for the male is 30, for the female, 40 grams. Lowered function of the posterior lobe causes a notable increase in this level, while hyperactivity may lower the so-called threshold to the point where a continuous melliturla ensues. The thyroid is influenced in the same sense but in minor degree, the averages closely approximating the normal levels. With lowered gonad activity in the female, the threshold drops to about half the normal level; with the male there is no change. Hyperactivity has never been observed by the writer. Adrenal results are anomalous and this point will be touched upon later. In the sex difference, the writer sees the potential influence of mammary function. The female has a mechanism for synthesizing, storing and conjugating galac-

tose of which the male is devoid. The problem is now under investigation but certain well known facts can be adduced to support the theory. The lowered threshold after the menopause and the mellituria of pregnancy are both observations pertinent to the question. Surgical ablation of the ovaries produces a drop in the sugar tolerance, of the testicles causes no change. In the two tests, the Basal Metabolic Rate and the Galactose Tolerance, is found the basis for a differential diagnosis between Gonad, Pituitary and Thyroid. Assuming lowered activity, the several findings can best be shown in tabular form.

TABLE XV

	Endocrine Focus		
	Thyroid	Pituitary	Gonad
Basal Metabolism	— — —	—	—
	+ + +	+ + +	+ + +
Sugar Tolerance	+	+ + +	Female — —
			Male 0

With hyperfunction of the two first the signs are reversed. The writer has never seen a proven case of hypergonadism. The adrenal finds no place in the scheme as indicated. The writer has seen too few genuine cases of adrenal disease to warrant any conclusions. A number of suspected cases submitted for examination have proven not to be adrenal conditions but have ranged from bronzed diabetes to primary aenemia. At present, adrenal cases can be evaluated only by exclusion but work is being actively prosecuted along several lines in the hope of establishing criteria for direct determination.

Similarly, several other of the endocrine entities have not been touched upon. Some of them have but tenuous claim to endocrine function, others are so rarely met with that at present data are lacking. The principle points of this paper may be summarized as follows:

SUMMARY

1. An objective method for the diagnosis of endocrine disorders is briefly outlined.
2. The results obtained in a series of 160 cases are statistically presented.
3. The thyroid influences the Basal Metabolic Rate profoundly, the Pituitary, Gonad and Adrenal only slightly but in the same sense.
4. Endocrine disorders tend to lower Blood Pressure.
5. Vital Capacities are least influenced by Pituitary disease; most by Thyroid.
6. The Dreyer standards based on chest and weight measurements are criticised adversely.
7. Thyroid cases apparently show lowered urine elimination, which is duplicated by the Phenol Sulphone Phthalein test. With this latter, the Adrental cases show the same tendency.
8. An appreciable number of Pituitary cases give a positive response to Ehrlich's "Urobilinogen" test.
9. Endocrine cases in general show a marked increase in the undetermined N_2 fraction in the urine.
10. The blood uric acid is raised above normal in many Pituitary cases without an equivalent increase in the other nitrogenous constituents.
11. Blood sugar levels are usually low in endocrine conditions.
12. Over 20% of all cases in this series show a low-grade mellituria. Only two of these were diabetics. Among the non-endocrine cases, syphilis and lesions of the central nervous system seemed to be predominant causes.
13. The majority of the endocrine cases show a marked lymphocytosis and an appreciable number demonstrate a mild eosinophilia. The gonad cases are an unique exception, their percentages approximating those of the non-endocrine cases.
14. The sex difference observed in the threshold of tolerance for galactose is noted and an explanation based upon diverse mammary function is offered.
15. The Pituitary exercises a profound influence on the carbohydrate metabolism; the Thyroid has slight effect and one in the same sense; the Gonad an intermediate effect, exercised in the opposite sense with women, and producing no change from the normal with men.

In conclusion, the writer wishes to express his indebtedness to his colleagues and his appreciation of the fine spirit of coöperation that they have so unfailingly shown. Their generous assistance and skilled service have done much to make these studies possible. To Doctors Babcock, J. Chandler, E. E. Chandler, Drury, Ellsworth, Fuller, Garrick, Greene, Hooker, Rowland, C. Smith and Wiggin, and to the laboratory staff of the Evans the grateful thanks of the author is gladly tendered. Especially is he indebted to his associate, Dr. C. H. Lawrence, who has shared in the latter stages of the work and under whose highly skilled and wise direction is carried out the subsequent clinical conduct of many of the cases.

THE INFLUENCE OF INTERNAL SECRETIONS ON GROWTH AND FUNCTION

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DURING the growth period, the power to compensate for derangement of normal processes is so great that it is extremely difficult to estimate the ultimate effect of any disease. It is only when growth is finished, or nearly finished, that the permanent effects of abnormal processes can be clearly seen, and it is then usually found that

the bodily machine has compensated for earlier damage far more completely than the most optimistic could have foreseen.

The degree of compensation, however, depends upon the amount of interference with nutrition which has continued to exist as the result of the primary pathological process. Whether or no such interference persists after the primary process is wiped out depends upon the effect of that process upon metabolism and the organs which control the various metabolic activities. As our knowledge increases, it becomes more and more apparent that those activities are controlled, to a great extent, by the internal secretions. Such conditions as diabetes, cretinism, acromegaly, and the cessation of menstruation following oöphorectomy leave no doubt of the enormously important influence of the endocrines upon nutrition, growth, and function, even if we are still ignorant of the exact mechanism through which that influence is exerted. Our ignorance is in large part due to the fact that conditions which we can recognize as due to derangements of internal secretion are usually the end results of such derangements. The changes which have taken place have become fixed, and nutrition and function can no longer be restored to normal, even if the causative influence be corrected. Because of our inability to identify malfunctions of internal secretions in their earlier stages, we are prevented from understanding the successive changes to which the end results are due, and from watching the stages through which the metabolism returns to normal. Therefore we are obliged to fall back upon theory, and great is the fall. Only when we are able to recognize the earlier stages of endocrine malfunction shall we come into possession of facts with which to replace theories, and thus come to a clear conception of the exact place of the internal secretions in bodily economy.

Of the methods for obtaining the necessary facts, the most important is, I believe, careful clinical study, checked by accurate objective findings. The constant coexistence of a certain derangement of growth, nutrition, or function, and a definite alteration or group of alterations in metabolism in the human body, is strong proof of the casual relation between the latter and the former. The return to normal in growth, nutrition, or function occurring as the result of the administration of gland extracts furnishes the final link in the chain of evidence.

In the hope of establishing some facts concerning endocrine function, we have been carrying on during the past year, at the Evans Memorial, a clinical and laboratory study of patients having functional or nutritional derangements of obscure etiology. In each case, a careful examination of the patient was made to exclude "organic disease" as the term is ordinarily understood. This included the history, physical examination, chemical and microscopical examination of the blood and urine, with such special examinations by x-ray or other methods as seemed indicated. The possible existence of unrecognised "organic disease" in these patients can, therefore, be pretty safely excluded. In connection with the clinical study, the patient's metabolic activity was determined by the methods described by Rowe[1]. When the evidence was all collected, the clinical and laboratory findings were correlated. On the basis of such a study, a working diagnosis was established, and treatment instituted. From time to time the patients returned to report their condition, and to have repeated such tests as would furnish objective evidence of change in their condition.

The patients so studied number 447. In 111, or 24.8% no evidence of endocrine malfunction was found. In 336, or 75.2% a diagnosis of derangement of endocrine function was established. Of these, 51, or 11.4% were due to thyroid disorder; 122 or 27.3% to pituitary dysfunction; 77 or 17.2% to gonad failure, and 8 or 1.8% to hypofunction of the adrenals. Polyglandular disturbance was found in 19 cases, or 4.3% of the total, and 59 cases, or 13.2% gave evidence suggesting endocrine disorder, but could not be classified. From the study of this series of cases, certain points seem reasonably clear. Perhaps the most important of these is the fact that clinical derangements of growth, nutrition, or function are accompanied by deviations from the normal metabolism which are characteristic of altered activity of some one endocrine function in the majority of cases. In less than 5% of all cases examined have we found it necessary to predicate a polyglandular dysfunction to explain the metabolic findings.

Although the metabolic alterations due to any given dysfunction are similar in every case, the clinical picture which they cause varies markedly according to the age at which the dysfunction became operative. Hypofunction of the thyroid gland produces three distinct types of individual, each type being apparently determined by the age at which failure occurred. Prenatal or congenital failure of the thyroid produces cretinism with its stunted physical growth and arrested mental development. Failure which becomes operative at or about puberty produces no marked arrest in growth or mental development, but interferes definitely with nutrition and efficiency. The resulting individual is usually below weight, deficient in energy, easily fatigued, and often exhibits depression or irritability of temper which may be due to malnutrition or to the consciousness of subefficiency. The clinical picture of myxedema is entirely lacking in this group, but is usually found in individuals in whom thyroid failure does not begin till growth has ceased. In each of these in direction and degree, the difference in the clinical pictures produced probably depending

groups the metabolic derangements are similar entirely upon the growth period in which the thyroid failure begins. Biedl[2] has called attention to the fact that the effect of administering endocrine extracts to animals varies considerably with their age.

The clinical pictures produced by pituitary dysfunction tend to vary in a similar way. The situation is however complicated by the fact that the pituitary consists of two lobes, and that dysfunction may be limited to either one or may consist in a derangement of both. Therefore the varying clinical pictures are not so clearly due to age influence. In a general way, however, prenatal or congenital failure produces dwarfism, with or without adiposity. The typical picture produced during adolescence is a slim, undersized body, though adiposity may be present and mask skeletal undergrowth. Failure during adult life may or may not produce adiposity, according to the lobe involved. Overactivity of the gland, which often precedes its failure, causes normal gigantism if it takes place early in life, acromegaly if it occurs after growth of the long bones has ceased. Yet whatever the clinical picture produced, the metabolic changes are the same for the same change in glandular function, whether it appear at birth, during youth, or in adult life. Deviation from this rule due to compensatory activity of some other secretion has not appeared in our series of cases. In those in which more than one secretion seemed to have been altered, the change in function was in the same direction in all the glands involved.

From our observations, it appears that the thyroid and the pituitary dominate growth and nutrition, especially in the male. This is not surprising, since between them they almost completely control the basal metabolism and the tolerance of the body tissue for sugar, and apparently have a very decided influence upon proteid metabolism. It is logical therefore that malfunction of either of these glands should have, as its result, the most marked derangements of growth and nutrition that the body can exhibit.

In contrast, the effect of gonadal dysfunction is less striking—and according to our observations, the changes in metabolism are less marked. Our studies upon the male castrate are too few to justify conclusions, but we have found, in those observed, very little effect upon general metabolism. All of our patients were, however, post pubertal castrates. It seems probable that when marked derangements of growth or nutrition are found in connection with hypofunction of the gonad in the male, the latter condition is an indication of, rather than the cause of the nutritional disturbance. The metabolic findings in secondary eunuchoid cases did not indicate gonadal influence. At present there appears to be no definite evidence that the male gonad does more than influence the development of primary and secondary sex characteristics, without having any marked effect upon general growth or nutrition. Wheelon[3] believes that the internal secretion of the testis acts as a differentiating factor, and that the withdrawal of this factor permits retrograde changes to occur in the characteristics dependant on gonadal influence. Our experience, limited as it is, suggests that the influence of the male gonad is limited to sexual characteristics and activities, and is not greatly concerned with general bodily development and nutrition.

In contrast, the female gonad apparently does have some effect upon the general metabolism, though in less degree than the thyroid or pituitary. Female castrates show a diminished sugar tolerance, and a tendency toward a lowered CO_2 exchange, similar to that in acidosis, but with no clinical symptoms of that condition. Clinically, the evidences of circulatory and nervous instability which we have found associated with gonad failure are evidences of the influence of the internal secretion of the ovary on the general bodily economy.

The successful function of the male gonad has but one purpose—the impregnation of the ovum. The successful activity of the ovary, however, predicates two functions: one of which governs the development which makes impregnation possible while the other is concerned with the results of this event. Some at least of the complex metabolic rearrangements incident to pregnancy are so similar to those found in ovarian dysfunction that it seems justifiable to ascribe them to gonadal influence. Therefore it is not surprising that in the female the gonad affects bodily nutrition and function more definitely than in the male. As a corollary, the symptoms of gonadal removal or failure are more marked in the former. Clinically ovarian failure produces instability of the circulatory and nervous systems, a tendency to moderate increase in weight, and often sexual frigidity or sterility, the picture being not dissimilar from that caused by the physiologic loss in function at the menopause. Women after the menopause likewise exhibit the same alteration in sugar tolerance that characterises pathological failure. What function the ovary has before puberty and what the effect of prepubertal ovarian failure might be, is not known.

One condition found in connection with ovarian, as well as pituitary and thyroid hypofunction is peculiar enough to deserve mention. We have examined 44 cases of otosclerosis for evidence of endocrine dysfunction, and have found definite evidence of metabolic derangement in 59%. Of these, 9, or 21% were typical of pituitary dysfunction; 3, or 7% of thyroid failure; and 6, or 14% were associated with definite evidence of ovarian hypofunction. The youngest patient in our series was ten years old, and in several the deafness appeared at about puberty.

The majority of the young patients had clinical symptoms suggestive of active endocrine disease.

Our experience with adrenal cases is too meager to justify definite conclusions. So far as they go they suggest that the adrenal does not play so important a part in the routine work of the bodily machine as do the thyroid and pituitary, and that its failure is expressed through an inability to mobilise energy rather than through nutritional or growth abnormalities. The few patients that we have studied, none of whom has had tuberculosis of the adrenals, have exhibited, as a characteristic symptom, profound depressions after relatively slight exertion, with subjective return to normal after sufficient rest. This complaint contrasts sharply with the constant sense of fatigue which is the outstanding feature in thyroid and pituitary failure. The definitely low blood sugar values which we have found in connection with adrenal failure suggest a possible basis for the rapid exhaustion. An enormous amount of study must be carried on before more can be said concerning adrenal function.

Concerning probable functions of other internal secretions, we have as yet no evidence.

It is not my purpose to present a statistical report in this article. Our patients were adults, and as I have pointed out, the symptoms caused by any given endocrine dysfunction vary according to the age at which it occurs. But since the histories in so many of our cases indicate that the dysfunction began in childhood or early youth, I wish to offer certain conclusions as to the role played by the endocrines during those age periods.

From our observations it seems evident that primary gonad or adrenal dysfunction rarely exist in the first decade. The rare cases which have been reported represent either hemaphroditism or precocious puberty. In contrast thyroid or pituitary dysfunction are much more common, and exercise a much more marked effect on general nutrition and growth. The clinical pictures produced by failures of these glands cannot always be distinguished by the history or physical examination, but simultaneous tests of vital function will usually show metabolic derangements which, in connection with the history and physical examination, establish a diagnosis. In children, clinical findings which arouse suspicion of endocrine dysfunction may be grouped as follows:

1. Abnormality of height or weight, without apparent cause, especially if either or both are not symmetrically distributed.
2. Departure from the orderly procedure of development, either physical or mental. The commonest evidences of such departure are abnormal closure of the fontanelles or epiphyses, and delayed dentition or puberty, and on the mental side, failure to display the intelligence average of the given age.
3. Extreme fatiguability, subnormal temperature, and bradycardia, if not explained by other findings, suggest an underlying metabolic derangement of endocrine origin.

It must always be borne in mind, however, that no one symptom makes the diagnosis, and that a child may be abnormal physically or mentally without disordered internal secretions. The signs enumerated above are suggestive. Only when they are confirmed by finding typical derangements of metabolism are they diagnostic of endocrine dysfunction.

With the onset of puberty such an existing dysfunction may disappear or may be increased. There is no way of knowing before hand what its course will be. In the female, latent hypothyroidism seems to became activated, for before puberty hypothyroidism affects male and female about equally, while after puberty the condition becomes six or seven times more common in females.

In as much as the lesser degrees of endocrine dysfunction may disappear at or soon after puberty, the advisability of endeavoring to treat them during the prepubertal period may be open to question. There can be no question, however, of the advisability of endeavoring to recognize them during the earliest possible period, for the earlier they are identified the more complete the restoration if treatment be deemed advisable. Moreover, only by such early identification shall we ever determine which conditions show exacerbations during puberty and therefore should be actively treated, and which tend to correct themselves and therefore need only be watched.

Another question which is raised in relation to treatment is whether or not any benefit is to be expected from glandular extracts except in thyroid failure. If the treatment is based on a correct diagnosis reached by correlating all obtainable evidence, and if it is carried out with the conviction which only such evidence can furnish, I believe benefit is to be expected. Unsatisfactory results may be as fairly laid to an improper diagnosis or an insufficient dosage as to the idea that the preparations are inert. In our experience results have been demonstrable with extracts of pituitary, ovary, and even adrenal cortex given orally. With these extracts the results were obtained, however, only when the amounts given were far larger than those ordinarily used. At present the dosage of any glandular preparation is perfectly indefinite, and will remain so until it is measured by units of active principle rather than by weight. The dose of any glandular preparation is the same as that of any other active drug, namely the amount required to produce the effect desired, if

the patient can tolerate such an amount.

In conclusion I wish to summarize the points which we believe to be important.

1. We believe that accurate diagnosis of disorders of internal secretion can be made in the majority of cases by combining certain vital function tests with carefully obtained history and a thorough physical examination.

2. We believe polyglandular syndromes form but a small fraction of endocrine disorders, and therefore that the use of polyglandular preparations, like polypharmacy in general, is illogical and useless.

3. Finally we believe that increase of our knowledge of the endocrine functions depends upon the collection of facts obtained by objectively controlled clinical studies.

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Reduction of Human Blood Sugar by Means of Insulin*

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FROM the beginning of our observations a definite relationship between the amount of insulin and the amount of urinary sugar excreted has been noted[1]. It was thought that there might possibly be a similar relationship demonstrable between the amount of insulin and the reduction of blood sugar. The recent literature[2 3 4 5] is quite uniform in holding that there is no such relationship. Attention has been called to the observation that in diabetics the amount of blood sugar reduction per given amount of insulin varies considerably in the same individual at different times. The following study has been carried out on patients showing blood sugars ranging from the normal level to marked hyperglycemia:

PROCEDURE

The cases are divided into three main classes:

Group I contains seven successive cases, all normal individuals having no symptoms of diabetes and with normal blood sugars.

Group II contains fourteen cases of diabetes. This group is further divided into three classes:
(a) Blood sugars ranging from .185 to .274 gms.
(b) Blood sugars ranging from .133 to .144 gms.
(c) Blood sugars ranging from .078 to .093 gms.

Some of these cases had received no insulin therapy prior to the experiment, others had been under insulin treatment for various periods of time (see Table II).

Group III contains twelve cases, all of which entered the hospital either in coma or in a precomatose state. This group is therefore subdivided into two classes: (a) coma cases, and (b) pre-coma cases. The blood sugars in class (a) of this group ranged from .213 to .757 gm. and the plasma carbon dioxide content from 10 to 26.5 (Cases 13-30). The blood sugars in class (b) ranged from .281 to .346 and the plasma carbon dioxide content from 15.2 to 18.6 (Cases 31-33.) The urine in all cases showed strongly positive ferric chloride reactions. (Two cases showing great cardiac failure and one complicated by toxic goiter are not included in this series.)

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The individuals in Group I were starved for twelve hours (the last meal being the 5 p. m. meal of the day before). The first sample of blood was taken about 6 a. m. and immediately thereafter an amount of insulin, sufficient to produce a definite and fairly marked reduction of blood sugar and yet not produce hypoglycemia, was injected subcutaneously. The individuals were kept at absolute rest in bed. Samples of blood were taken at 30 minutes, 60 minutes, 90 minutes, 180 minutes, 270 minutes and 360 minutes after the injection of the insulin.

The patients in Group II were observed in a similar manner; the period of observation extending over 180 minutes.

The patients in Group III were observed in the following manner: Upon admission samples of blood were taken for blood sugar and plasma carbon dioxide determinations, and from 20 to 50 units of insulin was injected subcutaneously immediately. In some cases another injection of insulin was given within the first hour or so. At the end of three hours another sample of blood was taken and again a known amount of insulin was injected. This was repeated in some cases at the end of six hours and again at the end of nine hours. In order not to cloud the effects of the insulin, other therapeutic measures were kept down to the minimum; only two cases (No. 22 and No. 26) received saline subpectorally; no food was allowed, and no intravenous glucose was given. Case No. 31 is the only one that had had any food within six hours prior to admission into the hospital. Case No. 22 received 11 gm. of carbohydrate about forty-five minutes after the initial sample was taken. Water was forced as soon as the patient could take it by mouth.

TECHNIQUE

Blood sugar determinations were done according to the Shaffer-Hartman method; plasma carbon dioxide content according to Van Slyke's method. The syringes used for insulin administration were the ordinary Luer syringes of 1, 2, 5 and 10 cc. volumes. The insulin used was of the H-10 (Lilly) standard, except in case 29 where U-10 was used but converted to H-10,

and case 23 where insulin prepared by the Department of Chemistry and standardized as H-10 was used.

RESULTS

Group I. Normal Cases. It will be seen from Chart I that in normal individuals the maximum fall in blood sugar for arbitrary doses of insulin takes place at about 90 minutes. Attention must be called to the parallel tendency of both the reduction and the recovery of the blood sugar. It is to be noted that the most rapid return to the normal level takes place in direct proportion to the weight in kilos per unit of insulin (or the amount of insulin per kilo of body weight). Take, for example, Cases No. 4 and No. 7. In case No. 4 one unit of insulin per 6.0 kilos body weight was used and the recovery in blood sugar was .005 gm. in the period from 90 to 180 minutes, while in case No. 7 one unit of insulin per 8.7 kilos body weight was used and the recovery in blood sugar was 0.10 gm. in the similar period. This corresponds to results which have been found in rabbits[6].

TABLE I

GROUP I—NORMAL INDIVIDUALS

Case	Wgt., kilo	Time, hr., min.	Blood sugar	Minutes elapsed	Reduction blood sugar	Insulin Units	Insulin Time, hr., min.
1	60	6:20	.0805			8	6 20 a. m.
(10867)		7:50	.061	90	.0195		
		9:20	.0725	180	.0080		
		10:50	.084	270	.0080		
		12:50		360			
2	67	6:15	.0685			9	6 15 a. m.
(10792)		7:45	.0545	90	.0140		
		9:15	.0625	180	.0060		
		10:45	.0700	270			
		12:15	.0685	360			
3	76	6:15	.0815			11	6 15 a. m.
(10898)		7:45	.0555	90	.0260		
		9:15	.069	180	.0125		
		10:45	.0815	270			
		12:15	.0830	360			
4	60.2	6:52	.082			10	6 52 a. m.
(10925)		7:22	.071	30	.011		
		7:52	.064	60	.018		
		8:22	.061	90	.021		
		8:52	.064	120	.018		
		9:52	.066	180	.016		
5	62	6:35	.088			8	6 35 a. m.
(15779S)		7:35	.059	60	.031		
		8:05	.067	90	.021		
		9:35	.079	120	.009		
6	61	6:30	.088			7	6 30 a. m.
(11302)		7:30	.079	60	.099		
		8:30	.074	90	.014		
		9:30	.084	120	.004		
7	44	6:25	.087			7	6 25 a. m.
(11286)		7:25	.056	60	.031		
		7:55	.070	90	.017		
		9:25	.078	180	.009		

Table I shows the patients' weights; initial blood sugars; amounts of insulin used and the blood sugar reductions at the times indicated. The total reduction in blood sugar ranged from .014 to .026 gm. with insulin dosage from 7 to 11 units.

From Table II it will be seen that, with the exception of case No. 2, the reduction in blood sugar per unit of insulin at the end of 90 minutes is very uniform, ranging from .0020 to .0026 gm.; an average of .0022 even if case No. 2 is included. In case No. 2 there was found a reduction in blood sugar per unit of insulin of only .0015 gm. It is to be noted that the initial blood sugar was .0685, whereas the lowest initial blood sugar of the other cases was .080 gm. This confirms the contention that the initial blood sugar level is a factor in the blood sugar reducing power of insulin[2] and [7], and will be discussed more fully later.

We will refer to the amount of blood sugar reduced per unit of insulin as R/U. Likewise the body weight in kilos per unit of insulin as W/U.

Group II, Diabetics. Here, as in the normal cases, there is noted a parallel tendency in the reduction of the blood sugars (Chart II). The maximum fall in the blood sugar takes place at about 180 minutes (Case 16), hence all calculations are based on the 180 minute period. As previously mentioned the cases have been divided into three groups:

Group (a) Initial blood sugars, ranging from .184 to .274 gm.

TABLE II

REDUCTION OF BLOOD SUGAR IN NORMAL INDIVIDUALS

Case	W/U	R/U
1 (10867)	7.5	.0024
2 (10792)	7.4	.0015
3 (10898)	6.9	.0023
4 (10925)	6.0	.0021
5 (15779S)	7.7	.0026
6 (11302)	8.7	.0020
7 (11286)	6.3	.0024
Average 90 minutes		.0022

R—Reduction in blood sugar.
U—Units of insulin.
W—Weight in kilos.

Group (b) Initial blood sugars, ranging from .133 to .144 gm.

Group (c) Initial blood sugars, ranging from .078 to .093 gm.

One is struck immediately by the difference in the amount of blood sugar reduction per unit of insulin of the averages of the three groups (Table IV), namely,

Group (a) .0094 gm.
Group (b) .0068 gm.
Group (c) .0033 gm.

that is, the higher the initial blood sugar level the greater the reduction in blood sugar per unit of insulin. The average amount of blood sugar reduced per unit of insulin (Group c) approximates that found in normal individuals and case 22 does fall within the upper limit of the normal values.

In diabetics, as well as in non-diabetics, the body weight in kilos per unit of insulin (W/U) is a prominent factor influencing the amount of blood sugar reduced. For example cases No. 9 and No. 10 (Table IV) have the same blood sugars and about the same glucose tolerance, but the W/U are 5.9 and 3.3 respectively and accordingly the R/U are .0066 and .0159 gm.

TABLE III

GROUP II—DIABETICS

Case No.	Wgt., kilo	Time, hr., min.	Blood sugar	Minutes elapsed	Reduction blood sugar	Insulin Units	Insulin Time, Hr., min.
8	60	8:35	.215			10	8:40
(10311)		11:40	.132	185	.083		
9	59	8:50	.245			10	9:00
(10296)		12:00	.179	190	.066		
10	32.7	8:35	.245			10	8:40
(10542)		11:45	.086	190	.159		
11	45	8:10	.207			10	8:12
(10644)		11:20	.124	190	.083		
12*	59	8:50	.207			10	8:54
(10623)		11:50	.094	175	.113		
13*	47	8:24	.184				
(10819)		10:00	.162	93	.022	10	8:27
		11:43	.127	196	.057		
14*	39.5	7:30	.259				
(10805)		9:30	.181	110	.078	10	7:41
		10:25	.159	165	.100		
15*							
(10827)	48.8	7:59	.198			10	8:00
		9:30	.166	90	.032		
		11:00	.104	180	.094		
16*	51	6:05	.093			8	6:05
(10717)		7:35	.070	90	.023		
		9:05	.0595	180	.0335		
		10:35	.057	270	.036		
		12:05	.0673	360	.0255		
17	41	6:30	.274			8	6:30
(11251)		8:00	.245	90	.029		
		9:30	.197	180	.077		
18	68	6:35	.142			8	6:35
(11234)		8:05	.109	90	.033		
		9:35	.078	180	.064		
19	43	6:00	.134			7	6:00
		7:30	.090	90	.044		
		9:00	.087	180	.047		
20	67	6:20	.133			6	6:30
		7:50	.103	90	.030		
		9:30	.093	180	.040		
21	70	6:30	.085			6	6:30
		8:00	.064	90	.021		
		9:30	.066	180	.019		

*Cases not having received insulin prior to experiment.

Again taking cases No. 18 and No. 19 whose blood sugars and tolerance are identical, the W/U are 6.1 and 9.5 respectively and the R/U are .0067 and .0057 gm. In this case the influence of weight, while present, is not as marked as in the first example.

A question that naturally arises is, what influence does the endogenous insulin production, which represents the tolerance of the patient, and probably is an index of glycogen storage, have on the blood sugar reduction brought about by exogenous administration of insulin? The possible effect of patients tolerance is illustrated as follows:

Cases No. 11 and No. 12 have the same blood sugars, while the W/U are 4.5 and 5.9 respectively. This would indicate that the R/U should be less in the latter. However, it is greater, and this may be explained by the fact that the tolerance of the former is 15 gm. glucose while that of the latter is 60 gm. Cases No. 13 and No. 15 may be compared in the same manner. Here the blood sugars have a difference of only .014 gm. and the W/U only .02, while the former has no tolerance for sugar and the latter has 47 gms. with a corresponding R/U of .0057 (the lowest in the group) and .0094 gm.

Thus it would seem that of the factors influencing blood sugar reduction, the initial blood sugar level is a primary factor. That weight is of great importance, is shown by the use of the rabbit weight in the standardization of in-

TABLE IV

REDUCTION OF BLOOD SUGAR IN DIABETICS

Case No.		Blood sugar	Tolerance	W/U	R/U
		Group (a)			
8	(10311)	.215	55 gm.	6.0	.0083
9	(10296)	.245	31 "	5.9	.0066
10	(10542)	.245	39 "	3.3	.0159
11	(10644)	.207	15 "	4.5	.0083
12*	(10623)	.207	60 "	5.0	.0113
13*	(10819)	.184	00	4.7	.0057
14*	(10805)	.259	40 "	4.0	.0100
15*	(10827)	.198	47 "	4.9	.0094
16*	(11251)	.274	55 "	5.1	.0096
				Average	.0094
		Group (b)			
17	(11234)	.142	55 gm.	8.5	.0080
18	(11251)	.134	112 "	6.1	.0067
19	(11311)	.133	112 "	9.5	.0057
				Average	.0068
		Group (c)			
20*	(10717)	.093	110 gm.	6.4	.0042
21	(11331)	.085	100 "	11.6	.0031
22	(11005)	.078	85 "	7.5	.0026
				Average	.0033

R—Reduction of blood sugar.
U—Units of insulin.
W—Weight in kilos.

*No insulin received prior to experiment. First four cases under active insulin treatment at time of experiment.

sulin, and by the above findings; that the endogenous insulin production plays some part we are convinced.

Group III. Cases in Coma. We have classified cases showing severe acidosis under two heads—coma and pre-coma states. The reason for this subdivision is that it was found that blood sugar reduction seemed to depend on the extent to which symptoms of acid poisoning had progressed.

TABLE V

GROUP III—COMA CASES

Case	Weight	Time Hr., min.	Blood sugar	Time Hr., min.	Reduction blood sugar	Insulin Units	Insulin Hr., min.	Remarks
23	31	2:45 p. m.	.580			30	2:45	
(10433)				1:15		23	4:00	1800 cc. saline
		6:35	.480	2:35	.100			
				1:25		17	8:00	
				2:00			10:00	10 gm. glucose
		11:50 p. m.	.263	1:50	.217			Gangrene left orbit
				9:05	.317	70		
24	45	3:15 p. m.	.757			50	3:15	
(10440)				1:05		50	4:20	
		7:15	.490	2:55	.267			
				3:00		50	10:15	Uncomplicated
		8:45 a. m.	.078	10:30	.412			
				17:30	.679	150		Dr. Shaffer's insulin as H-10
25	48.6	4:15 p. m.	.283			50	4:15	
(11225)		7:20	.202	3:05	.081	15	7:20	
		10:20	.130	3:00	.072	15	10:20	Uncomplicated
		1:15 a. m.	.097	3:00	.033			
				9:05	.186	80		
26	45	11:00 p. m.	.213					
(10923)		12:15 a. m.				20	1:15	(Hour late)
		3:00 a. m.		2:45		20		
		6:00 a. m.	.151	3:00	.062		3:00	Uncomplicated
				5:45	.062	40		
27	66	1:05 p. m.	.276					
(10950)		1:15		:10		30	1:15	
							2:30	2000 cc. saline
		4:15 p. m.	.248	3:00	.028			
				3:10	.028	30		Meningitis
28	54	8:15 a. m.	.345			40	8:20	
(11050)		11:20	.319	3:00	.026			
		12:15 p. m.		:55		20	12:15	
		3:30	.287	3:15	.032			
				7:10	.058	60		Meningitis
29	79	7:55 p. m.	.660			30	7:55	
(11284)		9:00		1:05		30	9:00	
		11:00	.544	2:00	.116	50	11:00	
		2:00 a. m.	.396	3:00	.148	30	2:00	
		3:00		1:00		10	3:00	
		5:00	.318	2:00	.078	20	5:00	Nephritis, chr. (mild)
		8:00	.286	3:00	.032			
				12:05	.374	170		
30	60	8:30 p. m.	.640			42	8:30	
(11752)		11:30	.610	3:00	.030	42	11:30	
		2:30 a. m.	.411	3:00	.199	70	2:30	Uncomplicated
		5:30	.342	3:00	.069	42	5:30	U-10 converted to H-10 units
		8:30	.253	3:00	.089			
				12:00	.387	196		

By coma is meant that state in which the patient is completely unconscious or, although responding to questions automatically, after recovery has a complete amnesia for the period of greatest acidosis. Patients in pre-comatose states may show the same plasma bicarbonate levels as those in coma, but respond readily to questions and have no amnesia.

Taking up the cases of coma, the more or less parallel tendency in the fall of the blood sugars

TABLE VI

REDUCTION OF BLOOD SUGAR IN COMA CASES

Case No.	Time	W/U	R/U	K	Remarks
23	3 hrs.	.58	.0019	.0011	CO_2 12.6 vol. %
(10433)	9 "	.44	.0045	.0019	29.6 — 57% increase
24	3 hrs.	.45	.0026	.0011	CO_2 14.5 vol. %
(10440)	17 "	.3	.0045	.0013	(17 hrs.)
25	3 hrs.	.97	.0016	.0015	CO_2 15.5 vol. %
(11225)	6 "	.75	.0023	.0017	(24-35% increase)
	9 "	.6	.0023	.0013	
26	6 hrs.	1.1	.0015	.0016	CO_2 26.5 vol. %
(10923)					CO_2 28.7 vol. %
27	3 hrs.	2.2	.0009	.0019	CO_2 38.1 vol. %
(10950)					CO_2 38.1 vol. %
28	3 hrs.	1.3	.00065	.00084	CO_2 13.5 vol. %
(11050)	6 "	.9	.00096	.00086	23-43% increased
29	3 hrs.	1.3	.0019	.0024	CO_2 10 vol. %
(11284)	6 "	.71	.0024	.0017	
	9 "	.52	.0022	.0011	
	12 "	.46	.0022	.0010	
30	3 hrs.	1.42	.00071	.0010	CO_2 15 vol. %
(11752)	6 "	.71	.0027	.0019	
	9 "	.39	.0019	.00074	
	12 "	.30	.0019	.00057	20.4 — 26% increased. 24—38% in 24 hrs. 17— at 36 hrs.

Averages

R/U				W/U × R/U			
3 hr.	6 hr.	9 hr.	12 hr.	3 hr.	6 hr.	9 hr.	12 hr.
.0016	.0014	.0015	.0011	.0015	.0014	.0015	.0011

(Case 11752 not included in above averages.)

will again be noted. Coma cases are comparable to diabetics in the matter of amount of insulin used, body weight, blood sugar level and tolerance. For example, comparing the W/U and the R/U for the three and six hour period (Table VI) it will be seen in each case that as the amount of insulin per kilo body weight increased, the amount of reduction in blood sugar increased. Again, there is no reason to believe that the initial blood sugar level should not influence the amount of reduction in blood sugar as it does in diabetics who do not show such severe acidosis, if one remembers that the saturation of the blood with sugar corresponds with the saturation of the body with sugar[3].

It occurred to us that because of the importance used in blood sugar reduction, these factors alone could be made use of to gage blood sugar reduction. It was found empirically in the first three cases that if the body weight in kilos per unit of insulin was multiplied by the reduction in blood sugar per unit of insulin that took place at the end of three hours a constant was derived that was strikingly similar. This expressed as a formula would be as follows:

$\frac{W}{U} \times \frac{R}{U}$ equals K (constant); and from this R equals $K \times \frac{U^2}{W}$

The average constant $\frac{WR}{U^2}$ of eight coma cases on which data was gathered to this purpose was .0015 (Table VI). Using R equals $K\frac{U^2}{W}$, the calculated blood sugar reductions did not vary more than 12 per cent from the actual observations. (Table IX). This holds true for both the three and six hour observations.

It may be that when more data is available this apparent uniformity in blood sugar reduction will be found to continue for a longer period of time, as the data at hand would suggest, provided the same manner of procedure is carried out.

Because two of the cases had meningitis, while a third had infectious gangrene of the left orbit, and yet showed this uniformity, it would seem that infection of itself does not reduce the blood sugar reducing power of insulin. However, attention is again called to the fact that in two cases with very marked cardiac failure and one case complicated by a toxic goiter this uniformity in reduction did not hold. Attention is also called to case No. 30 in which, if the late data (9-12 hours) may be compared, there was a relative impotency of the insulin, due possibly to the very marked depleted alkali reserve.

The results of $\frac{RW}{U^2}$ of those in pre-comatose state averaged three times greater than those in comatose state. (Table VIII).

DISCUSSION

It has been pointed out by Fletcher and Campbell that "with the lower blood sugars, at any rate, the low point most frequently occurs before four hours, and even with the higher blood sugars the point is not much delayed." The findings in this study bear this out, as regards the blood sugars of diabetics. It would seem that there is a difference between non-diabetics and diabetics in this respect, for the results herein given show the low point in non-diabetics to be reached about ninety minutes before that of diabetics. Wherefore, the results of this tance of the weight and the amount of insulin

study do not agree with their statement that "there is not any very evident difference between the effect observed in non-diabetics and diabetics." However, the results are in accord with their statement "it appears that in diabetics the blood sugar is easily reduced to the normal range by insulin, but somewhat less easily below this." In other words, there is a difference between the effect observed in diabetics and non-diabetics[7].

McCormick, McLeod, Noble and O'Brien pointed out that in rabbits, so far as the initial rate of the decline of blood sugar is concerned, a moderate dose has the same effect as an excessive one—that, we think, has been amply demonstrated by the charts, which show the striking parallelism in the rate of sugar reduction regardless of the amount of insulin used.

McCann, et al. have shown that in diabetics the amount of blood sugar reduction per given amount of insulin varies considerably at different times in the same individual; i. e., there is less reduction as the blood sugar mechanism approaches the normal.

Attention has already been called to the fact insulin is greater in diabetics than the normal, but greater in normal than in coma cases. An explanation that offers itself is as follows: The available glycogen or sugar in the normal individual is of such availability as to immediately offset any reduction in the blood sugar, this that the reduction in blood sugar per unit of taking place, probably through the attempts of the various internal secretory organs (adrenal, thyroid, pituitary, liver) to maintain the normal blood sugar level. In diabetics there is either a disturbed equilibrium of these internal secretions such that their actions are insufficient to replenish the blood with sugar, or, that the available supply of glycogen is of a considerably less amount than normal. In coma cases a different state of affairs exists, here we have a body saturated with sugar[8] so that any reduction in the sugar content of the blood is immediately replaced from the tissues of the body, with the result that the insulin per unit is able to reduce the sugar content only a small amount. By stretching this idea it may be surmised that the available sugar may be of such amounts that it can completely neutralize any blood sugar reducing power of insulin. This explains the case reported by Rabinowitz[4] in which no blood sugar reduction took place after fifty units of insulin. A similar observation was made on a case in the Barnes Hospital in which no blood sugar reduction took place after fifty units of insulin. However, this is of infrequent occurrence, as it happened only once in twenty-four observations on coma cases.

Though the human body cannot be used as a means for the assay of insulin, it should not be going too far to say that it should be possible to gage the amount of blood sugar reduction with some degree of accuracy when those factors, such as have been presented here, become more firmly established; at any rate the discrepancies should be explained in a plausible manner. It is

TABLE VII

PRE-COMA CASES

Case	Weight Kilo	Time Hr., min.	Blood sugar	Time Hr., min.	Reduction blood sugar	*Insulin* Units	Hr., min.	Remarks
31	58.6	9:30 p. m.	.281			20	9:45	Uncomplicated
(11005)		12:30 a. m.	.184	2:45	.097			
32	53	5:15 p. m.	.346			20	5:25	
(11115)		8:25	.282	3:00	.064	20	8:25	Uncomplicated
		11:25	.191	3:00	.155			
				6:00	.219	40		
33	53	4:00 p. m.	.268			28	4:10	Uncomplicated
(11714)		7:10	.107	3:00	.161			U-10 converted into H-10

TABLE VIII

REDUCTION OF BLOOD SUGAR IN PRE-COMA CASES

Case No.	Time	W/U	R/U	K	Remarks
31	3 hrs.	2.9	.0048	.0139	CO_2 15.2 vol. %
(11005)	9 "				33 vol. % - 54% increased
32	3 hrs.	2.6	.0032	.0083	CO_2 18.6 vol. %
(11115)	6 "	1.3	.0077	.0100	39 vol. % - 52% increased
33	3 hrs.	1.8	.0057	.0102	CO_2 17.4 vol. %
11714					
Average for 3 hrs.			.0045	.0108	

TABLE IX

CALCULATED BLOOD SUGAR REDUCTIONS

Case			
	Three Hours		
28 (11050)	$\frac{.0015 \times 40^2}{54} = .044$	Initial blood sugar	.345 .044
		Estimated	.301
		Actual blood sugar	.319
27 (10950)	$\frac{.0015 \times 30^2}{66} = .020$	Initial blood sugar	.276 .020
		Estimated	.256
		Actual blood sugar	.248
29 (11284)	$\frac{.0015 \times 60^2}{79} = .068$	Initial blood sugar	.660 .068
		Estimated	.592
		Actual blood sugar	.544
30 (11752)	$\frac{.0015 \times 42^2}{60} = .044$	Initial blood sugar	.640 .044
		Estimated	.596
		Actual blood sugar	.610
	Six Hours		
26 (10923)	$\frac{.0014 \times 40^2}{45} = .050$	Initial blood sugar	.213 .050
		Estimated	.163
		Actual blood sugar	.151
28 (11050)	$\frac{.0014 \times 60^2}{54} = .093$	Initial blood sugar	.345 .093
		Estimated	.252
		Actual blood sugar	.287
29 (11284)	$\frac{.0014 \times 110^2}{79} = .214$	Initial blood sugar	.660 .214
		Estimated	.446
		Actual blood sugar	.396
30	$\frac{.0014 \times 84^2}{60} = .165$	Initial blood sugar	.640 .165
		Estimated	.475
		Actual blood sugar	.411

of considerable clinical importance if insulin, as it is standardized at the present time, will produce a quite uniform reduction in the blood sugars of selected coma cases.

CONCLUSIONS

1. The reduction of blood sugar in normal individuals at rest per unit of insulin is strikingly similar. The blood sugar level is a factor in the amount of blood sugar reduction. The weight probably governs the duration of the effect of insulin.

2. The variations in reduction of blood sugar in diabetics may be accounted for to a great extent by the amount of insulin used per kilo of body weight, by the blood sugar level, and the amount of available body sugar.

3. Insulin produces a greater fall in the blood sugar of diabetics than in normals; the least fall in blood sugar of diabetics takes place in those cases whose blood sugar level approaches the normal, and may be as low as normal.

4. The least reduction of blood sugar per unit of insulin takes place in diabetic coma; the next lowest in pre-coma cases. The power of the body to restore the alkali reserve, marked cardiac failure and toxic goiters probably influence the action of insulin.

TABLE X

PRE-COMA CASES

CALCULATED BLOOD SUGAR REDUCTIONS

Case			
	Three Hours		
31 (11005)	$\frac{.0108 \times 20^2}{53} = .081$	Initial blood sugar	.281 .081
		Estimated	.200
		Actual blood sugar	.184
32 (11115)	$\frac{.0108 \times 20^2}{58} = .074$	Initial blood sugar	.346 .074
		Estimated	.272
		Actual blood sugar	.282

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7 Note: Joslin, 3rd ed., 1923, p. 70-71, has also called attention to this.
8 Palmer: J. Bio. Chem., 30, 79, May, 1917.
Joslin, 3rd ed., p. 70.
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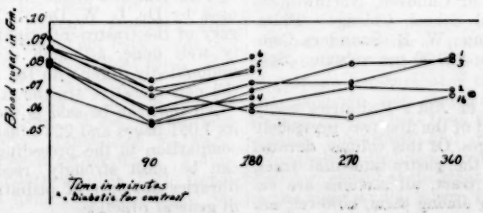
Chart I
Rate of Blood Sugar Reduction in Non-Diabetics



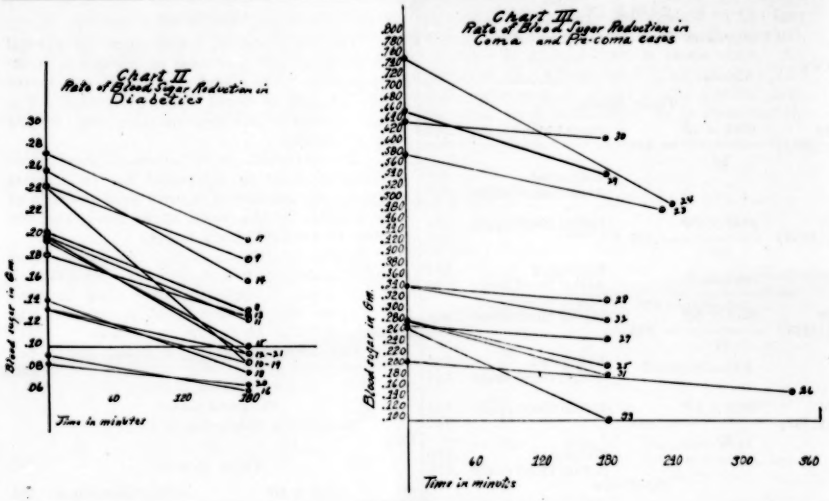
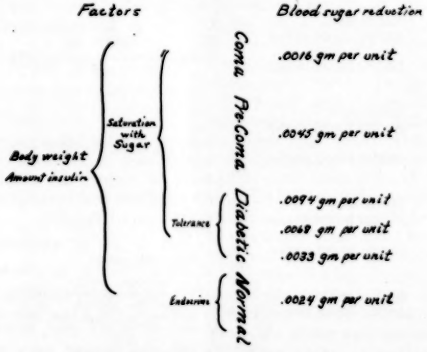


Chart IV



Schematic drawing showing blood sugar reduction and probable influencing factors

BOOK REVIEW

Abt's Pediatrics (Volume III). By 150 specialists. Edited by Isaac A. Abt, M. D., Professor of Diseases of Children, Northwestern University Medical School, Chicago. Philadelphia and London: W. B. Saunders Company, 1924. Cloth, $10.00 per volume. Sold by subscription.

The third volume of Abt's Pediatrics easily keeps up the standard of the first two, previously reviewed in these pages. Of this volume, devoted largely to surgery, the gastro-intestinal tract, and the respiratory tract, all sections are excellent. Outstanding among them, however, are the sections on Harelip, Cleft-palate and Allied Malformations, by Dr. James S. Stone; Nutritional Disturbances of Infancy, by Dr. Isaac A. Abt; Ciliac Disease, by Dr. McKim Marriott, and the Diseases of the Nose and Paranasal Sinuses, by Dr. L. W. Dean. The section on Surgery of the Gastro-intestinal Tract is exceedingly well done, although some surgeons would undoubtedly differ with Dr. Richter, who prefers the non-operative treatment of inguinal hernia.

All that can be said is that this volume, with its 1,051 pages and 223 illustrations, is a worthy comparison to the preceding volumes. The set can be most strongly recommended for the libraries not only of pediatricians, but of those in general practice.

Case Records
of the
Massachusetts General Hospital

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICO-PATHOLOGICAL EXERCISES

EDITED BY
RICHARD C. CABOT, M.D., AND HUGH CABOT, M.D.
F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 10301

An Italian-American boy of eleven entered March 19 complaining of anemia of four months' duration.

F. H. Good.

P. H. He had had no illness except measles at two years.

P. I. The middle of December he lost appetite, tired easily, and was less playful than usual. A physician who was attending his father for "rheumatism" gave the patient two injections in the arm. After the second the child became feverish. The doctor made a diagnosis of scarlet fever and kept the boy in bed for six weeks. There was no history of a rash, but about three weeks ago, six weeks after the diagnosis was made, the skin of the feet peeled. Since this attack he had been worse than before, with poor appetite and constipation, his bowels not moving for two or three days at a time. Enemata always gave good results. The urine was dark colored. He had occasional fever at night. For the past three weeks he had had "pain all over." His arms and legs were slightly tender. Motion was especially painful. For three weeks he had had slight unproductive cough, worse at night. In September he weighed 53 pounds, his best weight; present weight 49. He had been out of school since December 17.

P. E. A fairly well developed and nourished boy in some apparent pain. Anxious expression. Very pale. Mucosae of fair color. Tenderness on pressure over frontal and maxillary sinuses. Pea-sized glands at the angles of the jaw. Axillary and inguinal glands slightly enlarged. Marked Harrison's groove and rosary. Chest expansion greater on the right. *Lungs* hyperresonant throughout. Many musical râles, wheezes and crackles throughout both lungs, especially on inspiration. A few moist râles. Expiration prolonged. Tactile fremitus not increased. *Heart* not enlarged. Action regular and rapid. P_2 accentuated. A systolic murmur at the apex, slightly transmitted outward. Pulses normal. *Abdomen* rather tense. Voluntary spasm and tenderness over both upper quadrants. Liver a hand's breadth below the costal margin and very tender, not pulsating. Spleen (?) two cm. below the costal margin and tender. *Genitals.* Left testicle undescended. *Extremities, pupils* and *reflexes* normal. Hyperesthesia over the whole body, with questionable muscular tenderness.

T. constantly elevated and septic, only twice below 100° until the last five days of life and swinging daily to 104-105.8° during the first month, afterwards 102°-103.8°; terminal drop to 97° and rise to 104.6°. *P.* 80-180, usually above 120 and during the last week above 140. *R.* 20-60. *Urine.* Amount not recorded, sp. gr. 1.004-1.032, the slightest possible trace of albumin at three of twelve examinations, 2-3 leucocytes per high power field at one. *Blood.* Hgb. 60%, leucocytes 13,400-8,400, polynuclears 81-49%, a great many young polynuclears at the first examination, no immature forms four days later, reds 3,840,000-4,368,000, normal in size and shape, moderate achromia, platelets increased. *Wassermann* negative. *Widal* negative. Three *blood cultures* negative. *Sputum* negative at four examinations. Large diplococci and large bacilli at a fifth. *Stools.* Guaiac moderately positive at one of six examinations. *Pirquet* mildly positive. *X-ray March* 22. Heart shadow not unusual. . . Large areas of mottled dullness extending outward and downward from the right lung root along the course of the bronchi. . . . Plates of the antra not entirely satisfactory. *April* 4. Comparison with previous plates showed no definite change. From the appearance of the process in the right chest abscess could not be excluded. The recent plate, while not so good as the others because of respiratory motion, seemed to show increase in the clouding extending into the left upper chest, perhaps denoting extension of the pathology in this region. *April* 16. (See Plate II.) Very little change in pathological process in right lung since previous examination.

Orders. March 19. Fat free diet. Fluids not restricted. Soap suds enema in the morning. March 24. Syrup of hydriodic acid 3 i every four hours. March 26. Ferric citrate gr. 1½ every other day. March 27 and April 5. Ferric citrate gr. 1½. April 9. Saccharated ferric oxid gr. v by mouth 7 i. d. April 14. Sodium bromid gr. x. April 16. Chloral hydrate 20 drops by mouth. April 25. Adrenalin chlorid 5 drops s.c. p.r.n. for subnormal temperature and pulse of poor quality.

March 22 another examiner found no tenderness anywhere and believed the boy's complaint of pain on pressure over the head, extremities and abdomen was exaggerated. This interne found slight dullness from the angle of the right scapula downward and musical moist râles throughout both lungs, breath sounds harsh everywhere except at the right base posteriorly and in the right axilla, where they were softer and

the expiration was slightly prolonged. Below the angle of the right scapula at the end of expiration was a shower of crepitant râles. No heart murmurs were heard. The sounds were soft. The liver was not enlarged or tender, though low, perhaps because of marked grooving. The spleen was not felt, but the boy did not relax well. The lateral lower abdominal musculature was imperfect. Lateral to the lower recti there was ballooning outward of the aponeurosis.

The boy continued in about the same condition. The bowels continued to be constipated, moving only with catharsis or enemata. The cough was very slight and unproductive. By April 5 he had lost two pounds and seven ounces. April 8 there was dullness from the midback to the right posterior axillary line from the seventh rib to the base.

By April 24 there was more cough and expectoration. He still complained constantly of pain in the "stomach." There was more rigidity and tenderness over the right upper quadrant than over the other portions of the abdomen.

April 25 while sitting up the boy suddenly turned pale and his breathing became rather labored. The pulse was of poor quality; the rate could not be determined. The temperature was 97° by rectum. The skin felt cold and clammy.

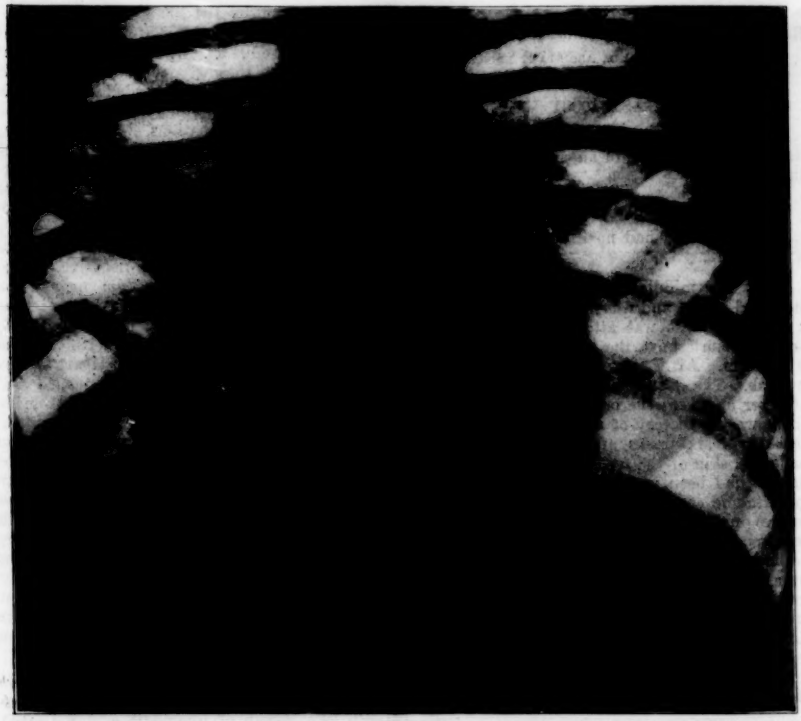


PLATE I. March 22. Large areas of mottled dullness extending outward and downward from the right lung root along the course of the bronchi.

Adrenalin gave relief. He had a very uncomfortable night, complaining of pain in the stomach, neck and head. The next morning while being attended to by the nurse he complained that suddenly everything turned black and he became dizzy. An hour and a half later he died.

Discussion

by dr. richard c. cabot

notes on the history

I cannot say just what those injections were for. What did he give him?

A Physician: Iron, for anemia.

DR. CABOT: Iron subcutaneously. I suppose that is it.

A PHYSICIAN: He probably had an Italian doctor.

DR. CABOT: That is a good point. It is common in Italy and on the continent everywhere to give it this way. It impresses the patient, but it is usually much better to give it by mouth.

NOTES ON THE PHYSICAL EXAMINATION

I have no idea what is wrong with the chest. There are râles in various places. There is said to be hyperresonance, which is so rare in a child that I do not believe it.

The pulmonic second is always accentuated at eleven, so I should discount that.

PLATE II. April 16. Very little change since the examination of April 4, when the report was, "Comparison with previous plates shows no definite change. The process is probably tuberculous. From the appearance of the process in the right chest abscess cannot be excluded. The recent plate, while not so good as the others because of respiratory motion, seems to show increase in the clouding extending into the left upper chest, perhaps denoting extension of the pathology in this region."

A PHYSICIAN: He got the scarlet fever from the injection?

DR. CABOT: He had hard luck. I don't believe the injection did it. And I do not see how we can call it scarlet fever merely on the fact that his feet peeled.

I cannot get any definite idea from the history. He seems to have had some fever, some general pains and general soreness, but it means nothing to me so far. If we did not have the physical examination I should be entirely astray.

In the heart there is what I should call a functional murmur. We shall have a chance to see. Perhaps there is something wrong here.

"Liver a hand's breadth below the costal margin" is the first definite point we have.

Has the undescended left testicle anything to do with the case?

A PHYSICIAN: A retained testis is likely to develop sarcoma.

DR. CABOT: Yes. Here is a case where we cannot help thinking of malignant disease. Sar-

coma is one of the forms most common in childhood.

The chart shows a temperature ranging between 101° and 105°, with big swings,—what is properly called a septic temperature. The pulse is always above 120, 120 to 170; the respiration is also high, 30 to 40. There is about five weeks of constant high fever, high pulse and high respiration, and so far we have no idea of their cause.

Presumably he was incontinent from time to time and they could not collect the urine. This albumin means nothing. We have normal urine and normal kidneys so far as we can tell.

There is no disease of the blood except a slight secondary anemia, secondary to some other cause which we must try to find.

A PHYSICIAN: How do you recognize young polynuclears?

DR. CABOT: They show less lobulation of the nucleus. This is what Arneth was always after. I do not think it is worth enough to look for it myself.

With such a long fever they had to be thinking of typhoid. Hence the Widal test.

A PHYSICIAN: Suppose he had had an antityphoid inoculation?

DR. CABOT: We should have had a positive Widal and might have been deceived. But we should then have noticed that he had a high white count, which we never get with typhoid, and a pulse relatively low. We should have looked for the spleen and wondered about the big liver. I think in the end we should have decided that he did not have typhoid.

They were looking for miliary tuberculosis. This case could perfectly well have been one of miliary tuberculosis. It has just the signs which we often see. But they did not get bacilli. It still might be miliary tuberculosis in spite of the negative sputa. We do not know yet. The Pirquet means nothing. There is hardly anything but negative evidence before we get down to the X-ray.

In a child we can generally see the heart shadow very well. We see it quite sharply in the plate of March 22. (See Plate I.) I do not myself believe that there will be any cardiac abnormality from what we can see here. But the areas to the right of the heart I should think were abnormal.

A PHYSICIAN: Is that a thymus shadow?

DR. CABOT: I do not think we have enough to say thymus there. I could not.

In the second plate, taken nearer, the heart looks bigger. We see masses especially definite at the points on the right where we saw them before, and we do not see much more on the left side.

We have no history that suggests abscess. Of course this shadow is in the position where abscess is most often seen,—the lower right lung—the place which owing to the course of the bronchus foreign bodies are most apt to reach. But he has had no pneumonia, no tonsillectomy, has not fallen into the water and got water into his windpipe, so we have no reason to suspect abscess.

The ballooning of the aponeurosis cannot have anything to do with this condition, as I see it. That is a congenital defect.

A very slight unproductive cough does not sound at all like abscess.

The condition on April 25 sounds like a hemorrhage somewhere,—not in the brain, because there is no paralysis. It sounds like an internal hemorrhage.

I think they were guessing, as I am, about the diagnosis up to the end. We will look at the additional X-ray plate and then we shall have all the facts. In the plate taken April 16 (see Plate II) there is a good deal more trouble on the right side than there was before.

DIFFERENTIAL DIAGNOSIS

What have we here? We have a boy of eleven; that is the first thing. There is something in his lung, something that causes fever, something that does not make any characteristic change in the blood, merely an anemia, something that does not, so far as we can see, involve the heart. We are not sure of anything wrong with the liver, because although it seemed to be enlarged, later they thought that it was the deformity of the chest that made it show. They were not sure of anything in the spleen. Now what are the possibilities?

A PHYSICIAN: Sarcoma.

DR. CABOT: Let us take first a term as general as we can, and not commit ourselves until we have to: neoplasm.

A PHYSICIAN: Miliary tuberculosis.

DR. CABOT: That is possible, though I have never seen so much of it on one side and so little on the other.

A PHYSICIAN: Chronic bronchopneumonia.

DR. CABOT: I never knew it to kill anybody in this way.

A PHYSICIAN: Abscess.

DR. CABOT: I do not see how we can exclude that, even though he did not spit pus.

A PHYSICIAN: Abscess of the liver.

DR. CABOT: That is very rare in this part of the world except in connection with gall-stones, and he has had no history to suggest that. I do not believe there is any abscess of the liver.

For me it comes down to two possibilities: malignant disease and abscess. If malignant disease, the commonest form is malignant lymphoma, which is sometimes called Hodgkin's disease and sometimes called sarcoma; the lymph structure is the essential thing. It can be confined to the chest, as this was. It can produce high fever as this did. That is never to be forgotten. I have often heard people say, "This cannot be malignant disease, because there is fever." It is per-

fectly possible for a rapidly growing neoplasm to give high, continuous fever.

A PHYSICIAN: Could a neoplasm grow so quickly?

DR. CABOT: It can grow just as fast as that; especially in children they grow very fast.

A PHYSICIAN: Have you an X-ray report on that?

DR. CABOT: "The recent plate . . seemed to show increase in the clouding extending into the left upper chest, perhaps denoting extension of the pathology in this region."

A PHYSICIAN: Could that be the result of amebic dysentery?

DR. CABOT: It could, with abscess of the liver penetrating into the lung. But I never knew it to come on in this way without obvious large amounts of sputum. And it is not at all likely that he got an amebic dysentery either in Brooklyn or in Boston. I think we can rule it out. If he had been born in any of the places where they have amebic dysentery that would be perfectly possible.

A PHYSICIAN: How do you rule out miliary tuberculosis?

DR. CABOT: I don't think we can. The points that seem to me against it are the sharp localization of disease at the bottom rather than at the top. So far as I have observed miliary tuberculosis it is peppered in both lungs, as if one had fired a charge of shot. We often get negative sputum in miliary tuberculosis, so that is not enough to rule it out. But I have never seen a case with such X-ray appearances. I do not see many cases with good X-ray plates, but it will be a surprise and a new point to me if this is miliary tuberculosis.

A PHYSICIAN: Does the negative Wassermann rule out syphilis?

DR. CABOT: No, but we have no lesions and no history to make us suspect it, and almost every time I diagnose it they refuse to find it post-mortem.

A PHYSICIAN: Would it be advisable to put a needle into that chest?

DR. CABOT: I do not see why one should not put a needle in. I do not know that it would do any good. If I seriously thought he had pus I should put it in. But I do not seriously think he has pus.

A PHYSICIAN: With an abscess would you expect to find as much change in the left lung?

DR. CABOT: No, I should not. But I never knew an abscess of the size this would have to be that did not communicate with a bronchus, and if it communicated with a bronchus he would have to spit pus.

A PHYSICIAN: Would a lymphoma spread as far out as this?

DR. CABOT: Yes, so far as I know it would.

I am going to say neoplasm, and the commonest neoplasm is lymphoma, so I am going to say lymphoma, without any reasons for saying so besides statistical ones. But I still do not see why he died, because there are no pressure symptoms. Most of the cases that die have pressure symptoms. I suppose we have to say there was a great deal more neoplasm than showed, perhaps in the liver or elsewhere.

MISS PAINTER: The X-ray report on April 4 gave this conclusion: "The process is probably tuberculous. From the appearance of the process in the right chest abscess cannot be excluded."

DR. CABOT: The X-ray people think it is tuberculosis, and they are generally right. But I have committed myself the other way, and I am not going to take back track merely because I hear that report.

X-RAY INTERPRETATION APRIL FOURTH

The process is probably tuberculous. From the appearance of the process in the right chest abscess cannot be excluded.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Pulmonary tuberculosis.
Tuberculous meningitis.

DR. RICHARD C. CABOT'S DIAGNOSIS

Malignant lymphoma.

ANATOMICAL DIAGNOSIS

Tuberculosis of the bronchial, mediastinal, mesenteric and retroperitoneal glands.
Miliary tuberculosis of the lungs with caseous pneumonia.
Tuberculosis of the kidneys.
Tuberculous ulcer of the ileum.

DR. RICHARDSON: The skin and mucous membranes were very pale, the skin smooth, showing an underlying bluish tint. The abdomen was somewhat distended and to the touch felt as though there might be an enlarged liver.

The peritoneal cavity contained 100 c.c. of thin pale fluid. Just above the ileocecal valve there was a tuberculous ulcer, a very small one, two millimeters across. The intestines were otherwise negative.

The mesenteric and retroperitoneal glands were greatly enlarged. A mass of the latter under the liver pushed the liver forward and downward, so that the anterior margin of the liver on the right was nine cm. below the costal border and thirteen cm. below the ensiform, and on the left four cm. below the costal border. The diaphragm on the right was at the sixth rib, on the left at the sixth interspace,—both down.

We have evidence here of a boy of eleven whose thymus had disappeared.

There were great masses of caseous bronchial glands. They more or less encircled the primary bronchi and evidently constricted them to some extent. In the region of the root of the right

lung there were enlarged caseous glands, and in close association with them a large irregular caseous mass extended across the lung. In the lower lobe there were smaller similar caseous masses. In the apices of the lungs were tubercles and a few very small caseous areas. The tissue of this lung elsewhere showed scattered tubercles. The left lung showed miliary tuberculosis but only a few small caseous areas.

DR. CABOT: That process did not start at the apex, did it?

DR. RICHARDSON: Apparently not.

DR. CABOT: This is the first case I ever saw in which it did not.

DR. RICHARDSON: The liver was slightly enlarged. No definite tubercles were made out in the liver macroscopically or microscopically. Frequently in tissues where macroscopically we cannot find tubercles we may find them with the microscope. In the spleen there were no definite ones macroscopically, but some were found in the microscopical sections.

The kidneys showed a few scattered areas of tuberculosis, but were otherwise in good condition.

DR. CABOT: It is worth while, now that we know the truth, to come back to this plate which I thoroughly misinterpreted. I suppose we should say that this shadow is the glandular mass.

DR. RICHARDSON: Yes. The pictures illustrate well the rapid progress of the disease.

DR. CABOT: You said there was no fluid in the pleural cavity. With all that shows in this last plate there is no fluid. All of this shadow is what Dr. Richardson has been describing. The process is entirely within the lung,—not a bit in the pleura.

A PHYSICIAN: What caused his death?

DR. RICHARDSON: Tuberculosis.

A PHYSICIAN: Why was there no diarrhea?

DR. CABOT: Dr. Richardson's ulcer was not very big,—two millimeters in diameter. We do not expect an ulcer of that size to cause diarrhea. That is all he found. Where did this process start, Dr. Richardson?

DR. RICHARDSON: From the anatomical picture I should say in the glands.

DR. CABOT: It did not start in the gut and crawl up?

DR. RICHARDSON: Probably not.

DR. CABOT: Apparently then it started from the chest glands. I have said once or twice that tuberculosis does not start in the base of the lungs; it starts at the apex, and this is the first case that I can remember of proved tuberculosis starting at the base.

A PHYSICIAN: Didn't they use to teach us that in children it started at the base?

DR. CABOT: I think so. I do not think it is true.

A PHYSICIAN: Are those sharp moist râles pathognomic of tuberculosis or of malignancy?

DR. CABOT: I never knew it of malignancy. It started in the base of the lungs apparently, and the newest process was the miliary process at the apices. This was in the last few days or weeks I take it.

A PHYSICIAN: What is the normal leucocyte count in a child of that age?

DR. CABOT: Anywhere from 8,000 to 16,000.

A PHYSICIAN: From the symptoms quoted here do you think this boy might have died of cardiac failure?

DR. CABOT: No, I think he died of tuberculosis. He had tuberculosis, then he got more, and then he died.

DR. RICHARDSON: There is the question of the constriction of the bronchi by the encircling enlarged caseous glands.

DR. CABOT: Yes, you thought there was some compression.

CASE 10302

A nine-months-old girl of Italian descent entered May 2. The complaint was cough of eight days' duration.

F. H. Good. Her mother had had no other pregnancies.

P. H. She was born at full term, normal delivery, and was normal at birth. She was breast fed for two weeks, then fed by a formula, and gained regularly. At two weeks she suddenly became unable to open her eyes. She was taken to the Eye and Ear Infirmary, where she remained for five weeks. A clinical diagnosis of gonorrhea was made, although no positive smear from the eyes was ever obtained.

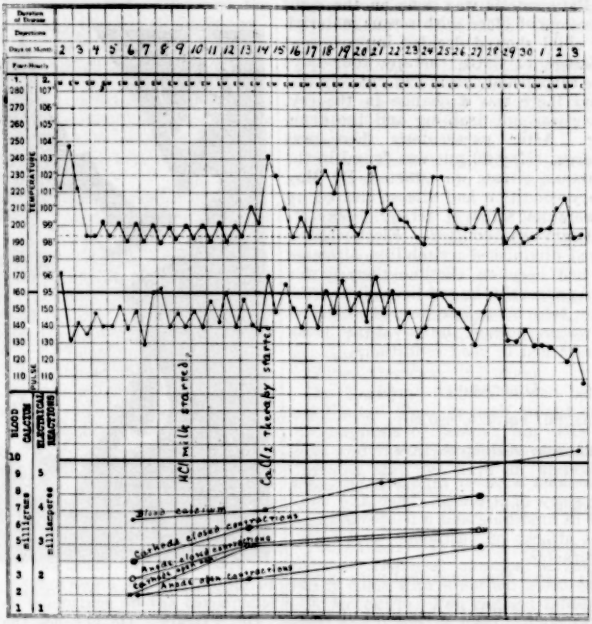
P. I. Two weeks before admission she had "bronchial asthma," more or less intermittent, the single attacks of difficult breathing lasting about half an hour. She was not cyanotic. She had a temperature of 101°. Eight days before admission she caught cold and had slight cough and some nasal discharge. This gradually improved. The morning of admission the mother noticed that the baby was breathing quite rapidly and held her hands rather stiffly. She had no spasms or convulsions. The local physician made a diagnosis of pneumonia. Throughout the illness she had taken her formula well, her bowels had been regular, and her pupils normal.

P. E. A well nourished baby with very rapid respiration and somewhat cyanotic skin and mucous membranes. Anterior fontanel admitted one finger. Eyes inflamed. Pharynx injected. Tonsils moderately enlarged. Palpable cervical and submaxillary glands. Moderate craniotabes. Moderate rosary. *Lungs.* Expiration prolonged. *Heart, abdomen, pupils,* and *reflexes* normal. *Extremities.* Carpopedal spasm.

T. and *P.* are shown in the chart. *R.* 90 at entrance, afterwards 36-68. *Urine.* Amount and sp. gr. not recorded, alkaline at four of seven examinations, a very slight trace to the slightest possible of albumin at three, 1-10 leucocytes per high power field, uncentrifuged, at five examinations; 75-100 leucocytes at the sixth examination, centrifuged; none at the seventh. *Blood.* Hgb. 70%, leucocytes 9,100, polynuclears 57%, reds 5,600,000, moderate variation in size, slight achromia. *Wassermann* and

"Tonsils of moderate size. Show no evidence of infection. Rather large amount of adenoid material in nasopharynx which in view of the aural condition of patient it would be better to remove."

Orders. May 2. Formula, whole milk ounces xviii, water ounces xviii, dextri-maltose 3 tablespoonfuls; six feedings of ounces vi each. Watch for cyanosis. Ounces vi of boiled water between feedings. May 9. Formula,



two *Pirquet* tests negative. *Stools* negative. *Blood phosphorus* 4.3 mgm. *Blood pH* 15 minutes after feeding (the child cried a little) 7.46 (normal 7.40-7.45). *Blood* CO_2 52.3 volumes per cent. *Blood calcium* and *electrical reactions* as shown in the chart. *Blood NaCl* May 8 4.88 mgm. per 100 c.c. (normal 5.5-6 mgm.), May 14 5.91 mgm. (four days after HCl milk started), May 21 6.03 mgm. (five days after $CaCl_2$ therapy started), June 3 5.95 mgm. (seven days after Alpine lamp and cod-liver oil therapy started). *X-ray.* Heart shadow apparently enlarged downward and to the left and prominent in the region of the pulmonary artery. Possibly congenital heart. Mottled dullness at both lung roots extending well outward and upward across the chest. These dull areas are quite dense and sharply defined. No evidence of enlarged thymus. *Throat consultation.*

whole milk ounces xviii, water ounces xiv, 1/10 normal HCl ounces iv, dextri-maltose 3 tablespoonfuls, six feedings of ounces vi each (milk should not taste sour). May 14. $CaCl_2$ gr. xv. Formula, whole milk ounces xxvii, water ounces xv, dextri-maltose 4 large tablespoonfuls, six feedings of ounces vi. Adrenalin chlorid 1/1000 solution one drop in each ear. Argyrol and adrenalin chlorid mixture 3 drops in each nostril every four hours. May 15. $CaCl_2$ gr. xv. t.i.d. May 18. $CaCl_2$ gr. xxv t.i.d. Dry wipes to right ear every four hours. May 21. Potassium citrate gr. xv t.i.d. Alpine lamp treatment and cod-liver oil.

About two hours after admission there was a sudden drop in respiration, the cheeks became flushed, the baby cried and looked much improved. The temperature rose to 103°, proba-

bly because of the fatigued condition. After a few hours it dropped to 100° and the general condition remained good. The following day the child was in good condition. The chart was normal. No lung pathology was found. Under treatment with HCl milk there was no apparent change in the patient's condition up to May 10. Three days later, however, she was definitely less irritable, the Chvostek sign could not be elicited, the carpopedal spasm was almost gone. No cause for the elevation of temperature May 14 was found except that both ear drums were injected. Paracentesis was done on the right with slight escape of blood with serous material. May 18 the child showed decided improvement since the change to $CaCl_2$ therapy four days earlier. The carpopedal spasm had entirely disappeared and there was a change in the cry. The right ear was not draining and the drum was bulging. After paracentesis there was serous discharge. May 20 pus was found in the urine. Potassium citrate was started. By May 24 the urinary condition had almost cleared up and there were no signs of tetany. May 27 potassium citrate was omitted. Electrical reactions showed diminished irritability, although the patient had been on alkaline therapy for five days. Apparently the cod-liver oil and the electrotherapy were enough to keep the calcium metabolism within normal limits. June 3 the child was discharged in good condition.

Discussion

by dr. fritz b. talbot

bedside demonstration

Bronchial asthma is rather rare in babies. We have a clear story of asthma. Then the baby had difficulty in breathing and a temperature of 101°, and the attacks of "asthma" were intermittent in character.

A Student: Isn't that consistent with laryngismus?

Dr. Talbot: Yes. What other kind of asthma do we have in babies?

Dr. Romberg: That due to anaphylaxis.

Dr. Talbot: That's the same as bronchial asthma. It rarely is seen during infancy and is nearly always associated with digestive symptoms, such as vomiting and diarrhea, or skin symptoms like urticaria and eczema. What else might it be?

A Student: Pressure from thymus.

Dr. Talbot: Thymic asthma. Do you know what is the characteristic history of thymic asthma?

Dr. Mary Wright: The symptoms of difficulty in breathing are rather evident.

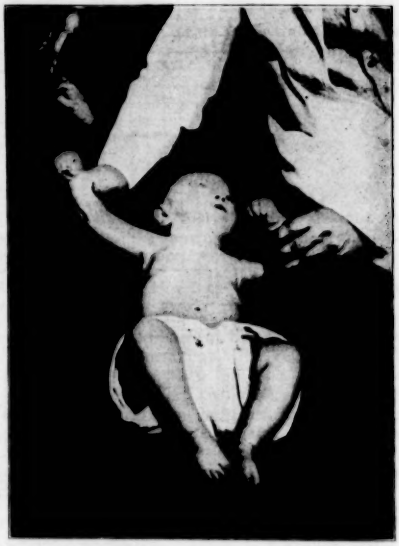
Dr. Talbot: Extension of the head backward may bring on the symptoms of thymic asthma. The attacks tend to occur at four o'clock in the morning. I have had cases that would come within thirty-five minutes of that time. It's a very interesting thing that it comes so regularly.

There is nothing abnormal in the blood. Can you draw any conclusions from that chart?

A Student: It shows that there is an infectious process.

Dr. Talbot: If there was one it is cured. What do you see about the baby that is abnormal?

A Student: There is bilateral carpopedal spasm. The thumbs are bent across the palm and the fingers are flexed. (See illustration.)



Shows the carpopedal spasm characteristic of tetany.

Dr. Talbot: Do you see the same thing in any other part of the body?

A Student: The feet are bent downward.

Dr. Talbot: We won't take time to go through a physical examination. The heart, lungs and abdomen are normal. Before we commence discussing this case we ought to know about the reflexes to see whether or not we are dealing with a neurological problem. The baby just did something that tells us. What did she do?

A Student: She turned her toes downward when I stroked the sole of her foot.

Another Student: She can grip. A person with a neurological lesion can't.

Dr. Talbot: Are the arms paralyzed?

A Student: No, they're moving.

Dr. Talbot: These are perfectly simple questions I am asking. What is it that might

make you think the hands are paralyzed? Has she opened up her thumb at all?

A STUDENT: No, she hasn't.

DR. TALBOT: Why may it not be a birth paralysis?

A STUDENT: It's bilateral, not unilateral.

DR. MCKENZIE: Cases of birth paralysis have flaccidity, not spasticity. This baby's arms are a little stiff.

DR. TALBOT: Is there any evidence of rickets?

A STUDENT: I am not very familiar with the normal, but I should say there is slight rosary.

DR. TALBOT: How about the wrists? Is there any enlargement of the epiphyses?

A STUDENT: I should say there is.

DR. TALBOT: Possibly a little. Where else do you look for signs of rickets? The frontal bosses are not enlarged. We have evidence of slight rickets and we have evidence here of tetany, the evidence being what?

A STUDENT: Carpopedal spasm.

DR. TALBOT: What are the other signs of tetany that we look for?

A STUDENT: Chvostek's sign.

DR. TALBOT: Yes, that is present. It is rather difficult to bring out. What is Chvostek's sign?

A STUDENT: Contraction of the facial muscles when you tap the facial nerve coming out from the anterior portion of the parotid.

DR. TALBOT: Are the reactions that I elicited normal in all babies?

A STUDENT: I think not in a baby of that age.

DR. TALBOT: In other words we can draw the conclusion that we have increased irritability of the nerve. What other sign is there besides the Chvostek?

A STUDENT: Trousseau's sign.

DR. TALBOT: What is the Trousseau sign?

A STUDENT: Compression of the middle of the upper arm or leg brings on the carpopedal spasm.

DR. TALBOT: How long does it take for it to come on?

A STUDENT: Three to four minutes.

DR. TALBOT: The quickest time it comes in is at least half a minute, and usually you have to wait for four or five minutes before you get it. Why don't we get it here?

A STUDENT: She already has it.

DR. TALBOT: What is the other sign of tetany?

A STUDENT: Erb's phenomenon, which is reaction of the muscles to a very low current.*

DR. TALBOT: What should the normal reaction be?

A STUDENT: I think it should be K O C.†

DR. TALBOT: How much lower does it have to go before it means tetany?

A STUDENT: Anything under five amperes, except for cathode closure contraction.

DR. TALBOT: How about the cathode opening of three?

A STUDENT: The anode opening is less than the anode closure. That is characteristic of tetany I think.

DR. TALBOT: What are the normal figures?

DR. KATZ: It varies with age, as I understand it. In a child five is the limit. But one examination is not sufficient, because there may be some variation from time to time.

DR. TALBOT: How about the cathodal opening?

A STUDENT: It is normal for an adult to have contraction of the low five.

DR. TALBOT: And here they were approximately 1.5 for the anodal opening, and for the closing 2.5.

Do you know what other findings we should have here? What do you expect in the spinal fluid?

A STUDENT: I think it should be normal.

DR. TALBOT: Yes. What do you find in the blood examination?

A STUDENT: Low calcium.

DR. TALBOT: What is the normal calcium?

A STUDENT: 10 mgm., I think.

DR. TALBOT: Yes, 10 to 12 mgm. How much was this?

HOUSE OFFICER: 6.5 mgm.

DR. TALBOT: So this is well below the normal limit. That in itself would make possible the diagnosis of tetany; but with the clinical signs and the Erb's phenomenon as well as the Chvostek sign we have an absolutely definite diagnosis of tetany.

A STUDENT: Is tetany associated with rickets?

DR. TALBOT: It is very commonly found with rickets. The next question of interest is, can tetany come with a normal blood calcium?

A STUDENT: You can get tetany from alkalosis and from a good many things besides rickets.

DR. TALBOT: In the infantile tetany we usually have the low blood calcium, I mean the characteristic infantile tetany connected with rickets; but we don't always have it, and the theory is that it is due to a change in the ionization of the calcium. What other forms of tetany are there besides this so-called infantile tetany?

A STUDENT: You can get it from pyloric stenosis.

DR. TALBOT: Gastric tetany, after operations.

A STUDENT: What causes it?

DR. TALBOT: During vomiting the chlorid is lost from the body and you have sodium left in excess, causing alkalosis.

A STUDENT: You can get it by rapid respiration.

DR. TALBOT: It does not come from rapid respiration alone. The Marathon runners

*Holmes, James B., The reliability of the electrical diagnosis of tetany. Am. Jour. Dis. Child., 1916, Vol. XII, p. 1.

†Cathode open contraction.

don't get it. But if you take a lot of CO_2 from the system by forced breathing or increased ventilation you get rid of the carbon dioxid and the alkalosis results. What is the third way?

A STUDENT: From food alkalies.

DR. TALBOT: What is the commonest alkali that is used?

A STUDENT: Sodium bicarbonate.

DR. TALBOT: May it be dangerous to feed bicarbonate of soda in large amounts?

A STUDENT: Yes; you have to be careful for several reasons.

DR. TALBOT: You may get tetany from the lowering of the calcium, from the change of the ionization of calcium, or from the normal calcium, and from increase of the alkali or decrease of acid ions in the body; and there again it all comes back to ionization of the calcium. The ionization of the calcium of adults is about .6 and that of the infant is .4. Anything below .4 is said to be accompanied by tetany. In other words the ionization of the calcium of the infant is just on the border line, so that any little thing which upsets the balance of the body towards the alkali side causes tetany.*

What other symptoms of tetany should we have if this had become worse?

DR. ROMBERG: Spasm of the larynx.

DR. TALBOT: What else?

A STUDENT: Pain.

DR. TALBOT: This baby doesn't like to be handled, but it is not so tender as others frequently are.

A STUDENT: Convulsions.

DR. TALBOT: Yes. How should we treat this baby?

A STUDENT: If you know it's tetany and that there is a low calcium of the blood it should be fed calcium.

DR. TALBOT: What other kind of treatment should we give? We may have a normal calcium. We may have an alkalosis.

A STUDENT: We should see that she gets some acid.

DR. TALBOT: We could feed calcium or acid or both. What form of calcium could we give?

A STUDENT: Calcium lactate or calcium chlorid.

DR. TALBOT: Why is calcium chlorid better than calcium lactate?

A STUDENT: It is not burned up as is the lactate in the body.

DR. TALBOT: What is it used for commercially?

A STUDENT: It is put on the streets to keep them from getting dusty. It absorbs water.

DR. TALBOT: Calcium chlorid has the virtue of supplying both calcium and acid. A baby may be given twenty-five grains three times a day. Is there any other way that we can treat this child than by giving calcium chlorid?

DR. ROMBERG: By hydrochloric acid milk.

*E. Freudenberg and P. György, Zur Pathogenese der Tetanie, Jahrbuch für Kinderheilkunde, 1921, Vol. 96, p. 5.

DR. TALBOT: We will start this baby in with twenty-five grains of calcium chlorid three times a day. I shall be very much surprised if the symptoms have not diminished markedly within forty-eight hours. If calcium chlorid is given and the symptoms disappear, will they return?

A STUDENT: I think if she is given a good milk diet by a proper formula they will stay normal.

DR. TALBOT: The chances are that they will come back again, so we shall have to do something that will prevent it. Since tetany is so commonly associated with rickets we can assume that some of the factors that are etiological in rickets may have an etiological connection with tetany.

That brings up the question as to tetany's being seasonal. The only answer that we can give at the present moment is that there is probably a connection between tetany and sunlight similar to that between rickets and sunlight. Tetany is said to have been cured with the ultra violet rays and with sunlight. You never see tetany in the summer and autumn. This baby must have sunlight. Cod-liver oil also acts well. The usual routine is to give calcium chlorid for four or five days, at the end of which time, if all the symptoms of tetany have disappeared, it is omitted and cod-liver oil is given as a preventive. As a rule the cod-liver oil prevents the recurrence.

NOTE BY DR. A. E. KOEHLER

This case shows very interestingly the disturbances that may result from an error in calcium metabolism. We have just had in the wards a case which offered a very similar picture except that there were no signs of rickets. This girl of seventeen had marked tetany following subtotal thyroidectomy with probable injury to the parathyroids. Her blood calcium was only half normal. What applied to her probably applies to this child—there existed a fundamental calcium metabolism disturbance so that the blood could carry only a subnormal amount. We have tried intravenous calcium chlorid with good but only temporary results. Calcium chlorid by mouth seems to be of value, but our results have been that it does not raise the blood calcium appreciably. Its beneficial effect seems to be due to increasing the acidity of the blood and hence producing calcium ionization. At one time when the patient who had been operated upon was in marked tetany we obtained complete relief by having her breathe ten per cent. carbon dioxid. It would be interesting to try this in a case like that of this child.

X-RAY INTERPRETATION

Definite pathology in the lungs of the bronchopneumonic type; but the character of the lesion is rather suggestive of tuberculosis.

DIAGNOSIS

Tetany, non-parathyroid.
Acute otitis media, suppurative.

CASE 10303

A German mill operative of sixty-two entered March 16.

At a previous entry seventeen years earlier radical cure of a small reducible left inguinal hernia of eleven days' duration was done. He made an uneventful convalescence, and a year later reported that he was in perfect condition, with no sign of recurrence.

F. H. His father and mother died long ago of unknown causes. He was not sure whether there had been tuberculosis in the family.

P. H. He had always been well except for an attack of "acute Bright's disease" fifteen years ago. (He gave the date of the hernia operation as fifteen years ago.) He had had recent buzzing in the ears. All his teeth were out. Several years ago he weighed 178 pounds, his best weight. His usual and present weight was 150.

P. I. A year ago he began to have dull constant pain in the left foot extending from the toes to the ankle, worse in the first two hours of the morning and at night after a day's work. Five months ago he noticed a crust forming on the lateral malleolus. This had since fallen off several times, to be replaced promptly by another crust. About this time he had callouses removed from the bottom of his left foot, as he had previously had done several times a year. Two weeks after this he had to give up work because the pain was so severe that he could not go back and forth. For three months he found that the foot got cold very easily, then swelled, turned cyanotic and was more painful. Four weeks ago a small black area appeared on the heel and extended rapidly. Three weeks ago the toes began to blacken, beginning with the second and in each case starting at the distal end. During recent weeks the pain had become so severe and throbbing that his doctor had to give morphia pills, as many as six a night. The only other procedure that gave the least relief was a constant change in the position of the foot, and this was only transient. He emphatically preferred death to continuing his present suffering.

P. E. A fairly well nourished old man lying with his left knee drawn up, not ill, but evidently in agonizing pain, although rather of the phlegmatic type. Mucous membranes cyanotic. Throat injected. Very little chest expansion. On deep respiration the lower left ribs flared markedly. Apex impulse of the *heart* not found. Measurements not recorded except left border 13 cm. from midsternum. Heart enlarged to percussion. Sounds poor. Pulses seemed of low volume and tension. B. P. 200/110. *Abdomen* and *genitals* negative. A truss effectively maintained a recurrent left inguinal hernia, reduced. Without the truss the hernia descended into the scrotum. *Extremities.* Fingers tremulous. Entire left foot swollen to a point two inches above the ankle. This region dusky red, and when blanched by pressure regained its color very slowly. All the second and fifth toes, most of the third, and part of the fourth were black and gangrenous. On the palmar aspect of the heel and on the lateral malleolus and the dorsum of the foot were other smaller areas. The foot was only slightly less warm than the rest of the leg. Sensation was virtually absent over the blackened areas. The rest of the skin on the left foot and all of that on the right was dry and scaly. Neither dorsalis pedis was felt. *Pupils* contracted. Right a little irregular. Reactions to light barely perceptible. *Reflexes.* Right knee-jerk normal; left could scarcely be elicited.

Before operation *T.* 98°-102°, *P.* 80-116, *R.* 20-31, amount of *urine* not recorded, cloudy at both of two examinations, sp. gr. 1.020-1.015, a slight trace of albumin and rare leucocytes at both. *Blood* not recorded. *Wassermann* negative. *X-ray.* Spur formation on the inferior surfaces of the os calcis. Roughening of the superior borders of the tarsus. Arteries of the dorsum of the foot visibly calcified. Tibial arteries visible, but not more so than in many other cases at this age.

The patient suffered considerable pain. A surgical consultant reported that he did not believe sympathectomy would relieve the pain sufficiently to warrant the procedure. Beginning March 16 the gangrene appeared to spread.

March 21 operation was done. The patient was not entirely rational next day. The temperature from this time ranged between 103.6° and 104.9° The pulse was 120, with a terminal drop to 50, the respirations 36, falling to 22. The blood pressure fell. By the 23rd there were a few râles in the bases and the patient was rapidly going downhill. March 24 he quietly died, apparently a cardiac death.

Discussion

BY DR. EDWARD L. YOUNG, JR.

This is the story of obliterative endarteritis of the lower leg and foot with the nutrition getting lowered to the point where it is unable to sustain the life of the tissues. This process can be a primary process, or can be secondary to chemical changes in the blood coming from diabetes or from nephritis, or it may be part of the general arteriosclerosis.

The treatment when this is starting is largely palliative, the attempt to increase the circulation of the foot by alternate hot and cold soaks, by dry heat and massage, by electrical stimulation.

A few drugs have at times been thought to help. Sodium citrate has been used recently, but there is no evidence that any drug has any real effect on the condition.

The procedure of arterio-sympathectomy has been tried for this on the basis that there is associated with this condition a spasm of the artery wall and that by cutting the fibers of the sympathetic nerve which run in the outer coat of the artery this spasm can be abolished. A number of cases have been tried throughout the country and reports vary, apparently depending on the amount of optimism of the operator. There is no real evidence that it is of definite benefit.

Cases such as this that have corns or callouses must be handled very carefully, as the tissue is particularly prone to sepsis, and when sepsis once starts it increases the danger to the patient very much. Whenever sepsis is present, particularly in the diabetic patient, a blood culture before operation will often give a better prognosis than the clinical appearance, because in many of these cases a slight sepsis will have a positive blood culture and will end fatally, and a clinically more advanced case may have no growth in the blood and will recover. In this case it seems as though the process had gone on so far that the only thing to do was to amputate the leg. One thing must be remembered, that in the occasional case even amputation will not entirely do away with the pain.

The anesthetic in these cases should be the one least likely to damage the patient, and for amputation that is spinal anesthesia. The question of the point of amputation is also important. Where the blood supply is as bad as in this case amputation below the knee is apt to be followed by either no healing or very slow healing of the stump with perhaps the need of further surgical treatment. So that amputation above the knee for the arteriosclerotic type of gangrene, such as this is, is the best procedure.

DR. YOUNG'S PRE-OPERATIVE DIAGNOSIS

Obliterative endarteritis of the lower leg and foot with gangrene.

PRE-OPERATIVE DIAGNOSIS

Arteriosclerotic gangrene of the left foot.

OPERATION

Under spinal anesthesia aided by gas-oxygen the left leg was amputated four inches above the knee-joint. After the femoral artery had been isolated and ligated elliptical flaps were made and sutured in place. There was a thrombosis in the femoral artery. Very slight bleeding from flaps. One cigarette drain. Ham splint applied.

PATHOLOGICAL REPORT

The upper end of the femoral artery is filled with a pale red plug.

A microscopic examination shows the walls of the artery degenerate and the lumen filled with organizing thrombus.

Arteriosclerosis.
Thrombosis.
H. F. HARTWELL.

FURTHER DISCUSSION

The patient did what he was entitled to do, went steadily down hill and died three days after operation. With the temperature I should expect there would be a positive blood culture at necropsy. Whether there will be other evidence of sepsis we have no right to state.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Arteriosclerotic gangrene of the left foot.
Chronic nephritis.
Operation, amputation of the left thigh.

DR. EDWARD L. YOUNG'S DIAGNOSIS

Arteriosclerotic gangrene of the left foot.
Thrombosis of the left femoral artery.
General septicemia.

ANATOMICAL DIAGNOSIS

1. *Primary fatal lesions*

Arteriosclerosis.
(Thrombosis.)
(Gangrene of the left foot.)

2. *Secondary or terminal lesions*

Amputation of left thigh.
Arteriosclerosis of the vessels of Willis.
Wet brain.
Fibrous myocarditis.
Hypertrophy and dilatation of the heart.
Arteriosclerotic degeneration of the kidneys.
Slight chronic pericarditis.
Slight hypertrophy of the spleen.

3. *Historical landmarks*

Ulcer of the duodenum.
Adenomata of the stomach.
Omental inguinal hernia (left).
Slight chronic pleuritis.
Obsolete tuberculosis of the bronchial glands and apices of the lungs.
Edema of the lungs.
Chronic splenitis.
Leukoplakia and erosion of the esophagus.

DR. RICHARDSON: Examination of the head showed a wet pia and brain. There was marked arteriosclerosis of the vessels of Willis which involved their remote branches.

The esophagus showed more or less leukoplakia with slight erosions. The stomach presented two very small adenomata, not cancer. The intestines were negative except for the presence in the duodenum at a point about 4 cm. be-

low the pylorus on the posterior wall of an old ulcer. The base of the ulcer rested over the head of the pancreas. There was but little reaction of the tissues in the region of the base, which was smooth and intact.

On the left side the internal abdominal ring admitted the passage of two fingers easily, and the canal contained a tag of great omentum which was easily removable except for a slender old adhesion to the wall of the upper part of the canal. There were only a few old pleural adhesions. The bronchial glands were slightly enlarged and several of them showed smaller and larger fibrocalcareous masses.

Lungs. In the region of each apex there was a small mass of obsolete tuberculosis. There were no areas of consolidation, but there was considerable edema.

Pericardium. There were several bands of old adhesions extending between the wall of the left ventricle and the parietal pericardium.

The heart weighed 510 grams, considerably enlarged. The myocardium generally was negative, except that in a few places in the wall of the left ventricle there were some areas of fibrous myocarditis. The cavities showed slight dilatation. The mitral valve showed a slight amount of the usual sclerosis. The aortic valve showed much sclerosis of the arteriosclerotic degeneration type. The coronaries showed marked fibrous and fibrocalcareous degeneration with some diminution of the lumen in places. The aorta and great branches showed well marked fibrous, fibrocalcareous and atheromatous arteriosclerosis. In the left femoral artery, which showed marked sclerosis, there was a small adhering thrombotic plaque. The pulmonary artery and its branches were large and the intima here and there showed small areas of fibrous sclerosis.

The liver, which weighed 1570 grams, showed a slight amount of congestion.

The spleen weighed 236 grams, slightly enlarged. Old adhesions bound the organ to the diaphragm. The tissue was rather soft with trabeculae, and the cut ends of the vessels showed fibrosis.

Kidneys. Left 142 grams, right 114 grams. The capsules stripped, leaving fairly smooth gray-brown-red surfaces which showed a few depressions in places. There was slight increase in the consistency of the tissue, and the cut ends of the vessels were rather prominent. The markings were fairly made out and the cortex measured 5 mm.

Culture from the heart blood showed no growth.

CONCENTRATED FOOD FOR THOUGHT

Mr. E. E. Rittenhouse of the Equitable Life Assurance Company in his report to the life insurance presidents says:

Most of all—no hurry and no worry.

CURRENT LITERATURE

ABSTRACTORS

GERARDO M. BALBONI — CHARLES D. LAWRENCE
WILLIAM B. BREED — TRACY MALLORY
LAURENCE D. CHAPIN — HERMAN A. OSGOOD
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PROHIBITION AND ALCOHOLIC MENTAL DISEASE

POLLOCK and FURBUSH (*Mental Hygiene*, Vol. VIII, No. 2, April, 1924) have made a careful analysis of statistics of the New York State Hospital Commission and the National Committee for Mental Hygiene in regard to alcoholic psychoses. The article gives in detail many tables of statistics. The conclusions are as follows:

Alcoholic insanity in this country is now much less prevalent than it was in 1910, but more prevalent than in 1920. The rate of decline since 1910 has been greater among women than among men. The reduction in alcoholic cases is due in part to a change in the habits of the people and in part to restrictive laws. The rate of alcoholic insanity is much higher among the foreign born than among the native white population. The rate is extremely low among native women of native parentage. The rate of alcoholic insanity is higher among negroes than among native whites. The enforcement of prohibitory laws is largely a matter of changing the more or less fixed habits of our foreign-born population. The rate of alcoholic insanity is much higher in cities than in rural districts. There is practically no alcoholic insanity among women in rural districts. Alcoholic insanity occurs principally in advanced middle life, following several years of excessive drinking. With respect to education, economic condition and marriage, patients with alcoholic insanity do not differ greatly from the general average adult population.

[H. R. V.]

PATHOLOGY OF RESPIRATORY EXCHANGE

LIEBESNY, from Denig's physiologic institute at Vienna, presents (*Wien. klin. Woch.*, May 15 and 22, 1924) his third communication on the pathology of respiratory exchange, dealing with the influence of iodine on metabolism. He finds that iodine in large doses has no characteristic effect on metabolism in persons without thyroid disease. In goitrous subjects with hypofunction, administration of iodine is beneficial, but not always so in cases with hyperfunction.

[R. M. G.]

AVIDITY OF ANTITOXINS AND THEIR CURATIVE VALUE

In a fourth communication (*Münch. med. Woch.*, March 14, 1924) KRAUS of Vienna reports the continuance of his experiments with scorpion serum on the significance of the avidity of antitoxins and their curative value. The neutralization power of an antitoxin is no measure of its curative power. There are antitoxins with curative properties, and others which possess no curative but only toxin-neutralizing properties.

[R. M. G.]

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THE TEACHING OF MEDICINE

THE teaching of medicine in the New World has had a distinctly different type of growth from that in the Old. Centuries-old established schools in Europe; the clinics of hospitals hoary with tradition were teaching the modern medicine of the day with methodical precision at a period when, in this country, the apprentice system was the only one by which practitioners were provided for large numbers of the population. The country was young; schools had not been established, and the trip to Europe was the only way in which an academic medical training could be acquired. Comparatively few were able to avail themselves of this instruction. A century and a quarter ago there were but three medical schools in the country, and only two general hospitals. Compare this situation with that at the beginning of the present century when, owing to the rapidity and the looseness of our national development, it was necessary to institute a vigorous weeding campaign in order that the selected and properly planted seedlings should not be overgrown by their undesirable neighbors.

Since Cos first became the Mecca of the afflicted; since the first healing herb was gathered; the practice, the study, the researches of medicine have had but one ultimate aim; the relief of sickness and the maintenance of health. In this it has been one of the practical sciences and one of the practical arts. Regardless of the individual desire to enter new realms of knowledge for the pure joy of discovery, the study and the investigations of medicine have been in the main practical. They have always; it is to be hoped they always will differ fundamentally from the developments in botany, in archaeology and in the fine arts. Osler said in the nineteenth century that "the great boon of this wonderful century, with which no other can be compared, is the fact that the leaves of the tree of science have been for the healing of the nations." Let the Oslers of our own century be able to pronounce the same.

The nineteenth century has left us a heritage of great achievements in our broad field. All of the fundamental sciences save anatomy have had their period of greatest development, if not, indeed, their conception, within its scope. The inspiration derived from its patient workers has been great, and under the hands and from the minds of many eager disciples their investigations have branched out like the branchings of a tree or of an artery. The domain of medicine can no longer be viewed by a single eye, or governed by a single mind. The physician can no longer be expected to court science in her laboratory, nor can the laboratory worker heal the sick. The science and the art are mutually dependent on each other, the one for the agency through which it works, and the other for the power it applies, but they are not one.

Part of our present difficulties in medical education are due to this separation of investigation and application—unfortunately necessary—and to the great strides that have been taken in increasing the total sum of knowledge in every field. Medicine, recently an infant, has suddenly outgrown its swaddling clothes, its nursery and its parents arms, and we do not quite know how to handle it; hence the stretching and the pulling of the medical curriculum, which, like the trolley car, must have a capacity limited only by infinity. This spatial difficulty is not at all helped by the extreme development of the clinical specialties, and it is becoming more and more important to ascertain, if possible, how much space should be allotted to each, and, in the small space allowed, how much should be taught.

The methods of teaching themselves are undergoing close and needed scrutiny. Which is of greater value, the didactic, or what we might designate as the stimulative method? The didactic method has certainly been the method of the past and we dare be reactionary enough to say that it has its good points if properly employed. At Glasgow a hundred lectures in Surgery, a hundred in Medicine and a hundred in Obstetries or Midwifery are still given. The minor specialties must take pot luck, as must

the students. We were told recently of a professor in that school who greatly prized his office safe; he kept his lectures in it and had for years. Perhaps it would have been better for the students, if more difficult for the professor, had the safe been rifled and a new set of lectures written perforce. A good lecturer, nevertheless, is of exceeding worth, for he is not only didactic but also stimulative. Such have been the Peppers, the Bigelows, the Holmeses and the Oslers.

Quite opposed to this method of training the mind to shoot is that of providing artificial vacua in the groaning curriculum; breathing spaces in which the student may rest, look about and think for himself—perhaps do a little research or other special work, or attend the ball games. A steady go in harness may not be desirable, but it is certainly equally questionable if the student, immature as he generally is, will hold true to the course when the reins are slackened. Research he may do in his spare time, but all are not Bests, and it is doubtful if his results will be more than trifling. The personal value of such work, however, must not be considered too lightly. The training derived from independent thought and the viewing of a problem from an investigative angle is certainly worth consideration.

Two types of individual have come to be necessary in the faculty of every school and on the staff of every hospital. So great has the complexity of our problems become, indeed, that each department must have its representatives of each type, both clinical and laboratory, and even a third; the administrative, and the administrator may be drawn from either basic group, for he may be found in either. It has come to be generally, but erroneously, believed quite lately that the head of a department or the chief of a service must be above all a laboratory man. We will grant that he should be an investigator, but we will not grant that the pendulum has swung so far away from the practice of the Art of medicine as to deny him the ability to investigate in the clinical field, to give adequate instruction to students, most of whom are to follow in his footsteps, or to organize and wisely direct a department in a school or a service in a hospital. It may, in fact, be wiser to have the clinician do much of the administrative work and most of the teaching; certainly as far as the latter is concerned he generally has a well-rounded experience that enables him to do it better, and the true researcher is thus spared the interruptions that will surely prevent him from doing well his chosen work.

The "Art" of medicine has been much discussed; the danger of its becoming a lost art has been frequently pointed out and this danger, if it exists—and who, under present conditions, can doubt it?—is a serious one. It has been said that such an art cannot be taught but must be acquired by slow and frequently painful experience. If any aid in this acquisition can be given, it must certainly come from those who have already acquired it, and this constitutes the greatest argument in favor of the part time teacher; not he who has reached his goal and retired from active participation, but he who is constantly letting down his bucket into the fresh current. That the Art cannot be taught we will agree, but its essence can be absorbed through personal contact; through contacts that emphasize the tremendous factor of personality in the selection of a teaching staff.

Each type of man, if he be representative of his type, is of value in the organization of a strong school of medicine, but care must be taken in his selection. There is danger of numerical overdevelopment—of having too many men, giving each too little time, under too little supervision. The part time practicing assistants are of great value for they may leave deep imprints upon their students, but they must be carefully chosen; they must be hand-picked, and they should be limited in numbers.

Research must be conducted in every school, in every hospital, in every department. Some contact between these cloistered workers and the students will be of benefit to both, and if they can teach and have the time for it let them do so, mindful of Osler's instruction that "you should, as students, become familiar with the methods by which advances in knowledge are made, and in the laboratory see clearly the paths the great masters have trodden, though you yourselves cannot walk therein."

Let us never forget, however, that the laboratory is an auxiliary to the main object of medicine—to care for the sick and to maintain the health of the sound. Let us never forget that great as is the need for sound investigation; invaluable as it is for the progress of medicine and for the advancement of our schools, the primary duty of these schools to their founders and to present and future generations is to train students, not for the laboratory—a few will seek this as water seeks its level—but for the practice of medicine.

THE WET-NURSE DIRECTORY

THE first attempt at establishing a wet-nurse directory in Boston was made over twenty years ago, consisting simply in a registry for nurses at the Boston Medical Library, on the same system as an intelligence office. This attempt failed for several reasons. In 1910 a second attempt, this time successful, was made by Dr. Fritz B. Talbot, and the present directory for wet-nurses was opened at the Hospital of the Massachusetts Infant Asylum in Jamaica Plain, funds for conducting the work having been privately obtained. Two worthy objects were furthered in this way—friendless mothers were cared for during a period after the birth of their babies; they were instructed in the proper care of their in-

fants and were given an opportunity of earning money, and an accessible source of breast milk was supplied which might be utilized for infants who were sorely in need of it, and who were deprived of the maternal source. Wet-nurses were carefully selected on the basis of health and milk production, and the directory was widely advertised in order that a demand might be created as well as a supply.

During the present year the Directory—now at 63 Binney Street, Brookline,—has greatly increased its facilities. The most recent innovation has been the addition to the staff of a social worker to collect milk which can be sold at a more reasonable price than has previously been possible. This milk is collected from healthy mothers in clean homes, brought to the Directory, pooled and pasteurized and sold to hospitals at twelve cents an ounce, or to private cases at twenty-five cents an ounce. The mother receives seven cents an ounce for her milk. In this way the supply of milk which has been obtained in the past at the Directory will be supplemented. The Infants' Hospital and the Massachusetts General Hospital have already provided funds for the purchase of the milk.

Six to eight wet-nurses live at the Directory with their babies. Health tests are required before admission, and the milk is sold as stated above, or if the entire supply of one nurse is needed for a case she is allowed to remain at the Directory at the charge of $30.00 a week. If she goes into the patient's home $20.00 a week is charged. Surplus milk has been given to deserving hospital cases, and in this way 10,000 ounces were given away last year. This, however, has not been entirely satisfactory from the hospital point of view because of the irregularity of the supply.

The Trustees of the Directory for Wet-Nurses, financed largely through private subscriptions, now wish so to increase their activities that breast milk may be supplied to all sick infants needing it in Boston and vicinity. Temporary funds are available for carrying on this work, but it cannot be continued unless its desirability is clearly shown by the demand for it. In other cities, notably Detroit and Cincinnati, the system of home collection of breast milk has met with great success; it should meet with the same success here, justifying the continuance of the experiment. If the demand is not created the expense will be too great to continue supplying milk at the present figure.

Every infant-caring hospital should, if possible, have a fund for the purchase of breast milk, either as needed or in fixed quantities. Every physician should realize the availability, through this philanthropy, of carefully collected and pasteurized breast milk. Only by a constant demand for the product, especially by hospitals, can it be shown that the efforts of the Directory are being appreciated, and their continuance guaranteed.

BURIAL

THE *London Medical Press and Circular* comments editorially and with slightly elegaic touch on the custom of burial. Food for thought is found in a visit to a large London cemetery, studded with enough marble to fill a quarry. In any large city the question of burial must be a serious one; in a city of the age of London this wasteful use of land must present many complications. We are bound by tradition in the disposal of our dead more, perhaps, than in any other of our terrestrial activities. When will we come to realize the waste of space and the waste of money in our efforts to perpetuate as long as possible that for which the laws of nature have prescribed return to dust?

The acceptance of cremation has been slow; it should be swift and universal. The painful costly pomp of funeral ceremony is a relic of the darker ages of man's intellectual outlook. It is a pitiful effort to hold that which has gone. Can we not recognize that if man lives after death he lives in the spirit, not in the body, and that the body without the spirit is not man?

"Were we to realize," says *The Medical Press*, "how a few centuries have sufficed to obliterate all evidence of the resting-place of the great majority of those who have gone before, we should more readily content ourselves with the assurance that the record of a life well-spent is inscribed on pages other than marble."

MISCELLANY

ATTENTION, FORMER ILLINOIS DOCTORS

WILL any and all doctors, former residents of Illinois, or descendants of pioneer physicians of the "Illinois country" communicate at once with the Committee on Medical History, Illinois State Medical Society, No. 6244 North Campbell Avenue, Chicago, Illinois?

Under the sponsorship of the Illinois State Medical Society there is in preparation "A History of Medical Practice in the State of Illinois" that must go to the printer at an early date. In order that this volume may be accurate and complete, all possible assistance is asked from every source, as to personal data and experiences, including diaries, photographs and similar documentary mementoes of pioneer Illinois doctors and of progressive phases of medical practice, as well as of achievements in fields other than those of medical science. Prompt return in good condition is promised for anything loaned the committee, the personnel of which is:

O. B. Will, M. D., Peoria, Ill.
C. B. Johnson, M. D., Champaign, Ill.
Carl E. Black, M. D., Jacksonville, Ill.
George A. Dicus, M. D., Streator, Ill.
James H. Hutton, M. D., Chicago, Ill.
Chas. J. Whalen, M. D., Chicago, Ill.—Chairman.

The scope of the volume will range from the discovery of Illinois to modern times. Through this period of over 250 years there is much of thrilling interest to be detailed. Collection of the human interest data can come only from the families or closest friends of the pioneers, many of whom long ago removed to distant sections of the United States. Through the kindness of editors of various medical journals, it is hoped to reach those who may be able to loan valuable material to the compilers who guarantee careful guardianship of anything sent for publication.

Some of the subjects touched will be: Physicians accompanying early explorers; government surgeons and physicians in attendance at the forts; early medicine in Illinois; theories of healing from the days of the Aborigines through the mound-builders; French and English explorers; the ante-boundary days; sporadic settlers; medical attendants for the covered wagon; herb doctors; primitive surgery; medicine and missionaries; migration of pioneer physicians to new territory; the "circuit-riding" and "saddle-bag" doctors and their burdens, triumphs and perils; pioneers as "utility citizens"; Illinois men in war time—there are four conflicts to be considered since the opening of the Nineteenth Century; Illinois medical men away from medicine, i. e., in industry, in science, in belles-lettres—art, music and literature.

Photographs especially are desired. Also copies of letters, statements of "cures" and "new methods," dairies and the like.

RHODE ISLAND STATE BOARD OF HEALTH

CONTAGIOUS DISEASES REPORTED TO THE STATE DEPARTMENT OF HEALTH OF RHODE ISLAND FOR THE WEEK ENDING JULY 5, 1924

Diphtheria	
Newport	3
Pawtucket	2
Providence	4
Warwick	1
Woonsocket	2
Scarlet Fever	
Pawtucket	2
Providence	7
Westerly	1
Measles	
Providence	5
Little Compton (German Measles)	1
Typhoid Fever	
Providence	1
Septic Sore Throat	
Cranston	1
Providence	1
Mumps	
Little Compton	1
Chickenpox	
Providence	1
Whooping Cough	
Providence	1

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

DISEASES REPORTED WEEK ENDING JULY 12, 1924

Disease	*No. of Cases*	*Disease*	*No. of Cases*
Anterior poliomyelitis	1	Ophthalmia neonatorum	14
Chickenpox	54	Pneumonia, lobar	37
Diphtheria	106	Scarlet fever	95
Dog-bite	12	Septic sore throat	1
Encephalitis lethargica	2	Smallpox	2
Epidemic cerebrospinal meningitis	1	Syphilis	36
German measles	9	Suppurative conjunctivitis	9
Gonorrhea	96	Trachoma	3
Hookworm	1	Trichinosis	1
Influenza	2	Tuberculosis, pulmonary	137
Malaria	3	Tuberculosis, other forms	28
Measles	245	Typhoid fever	12
Mumps	84	Whooping cough	44

CONNECTICUT DEPARTMENT OF HEALTH WEEKLY MORBIDITY REPORT

WEEK ENDING JULY 12, 1924

(Including all cases reported before 11 A. M., Monday, July 14, 1924)

Diphtheria	
Fairfield County	
Bridgeport	3
Danbury (T)	1
Greenwich	2
Shelton	2
Stamford (T)	1
Stamford (C)	1
Stratford	1
Hartford County	
Enfield	2
Hartford	6
New Britain	2
Litchfield County	
Salisbury	4
New Haven County	
Meriden (C)	1
Milford	1
New Haven	2
Seymour	1
Waterbury	4
New London County	
Colchester	1
Norwich (T)	1
State Total	36
Last week	19
The following diphtheria bacilli carriers were reported:	
Hartford	1
New Haven	4
Waterbury	1
Typhoid Fever	
Fairfield County	
Bridgeport	1
Hartford County	
Hartford	1
New Britain	1
Windsor Locks	1
New Haven County	
Hamden	1
Oxford	1
New London County	
Groton (B)	1
State total	7
Last week	1
Scarlet Fever	
Fairfield County	
Bridgeport	5
Danbury (C)	1
Stamford (C)	1
Hartford County	
Bristol	1
Canton	1
Hartford	6
New Britain	3
Plainville	1
Wethersfield	1
Litchfield County	
Thomaston	2
Watertown	4
New Haven County	
Hamden	8
Madison	1
New Haven	4
North Haven	1
Waterbury	2
New London County	
Stonington	1
State total	43
Last week	26
Smallpox	
Fairfield County	
Bridgeport	1
Windham County	
Brooklyn	1
Danielson	3
Plainfield	1
State total	6
Last week	15

Measles

Fairfield County	
Bridgeport	1
Sherman	1
Stamford (C)	3
Hartford County	
Enfield	1
Hartford	7
New Britain	1
West Hartford	7
Litchfield County	
Salisbury	12
Watertown	2
Middlesex County	
East Hampton	1
New Haven County	
Branford (B)	1
Madison	4
Milford	11
New Haven	5
North Haven	1
Woodbridge	2
New London County	
Groton (B)	4
North Stonington	1
Tolland County	
Hebron	2
Windham County	
Putnam (C)	3
Woodstock	1
State total	71
Last week	102

Whooping Cough

Fairfield County	
Bridgeport	3
Greenwich	5
Stamford (T)	1
Litchfield County	
Norfolk	6
Salisbury	2
Watertown	11
Middlesex County	
Killingworth	2
New Haven County	
New Haven	2
North Haven	1
New London County	
Preston	1
Windham County	
Pomfret	1
State total	35
Last week	23

Other Communicable Diseases

Cerebrospinal men.	1
Chickenpox	32
German measles	3
Malaria	1
Mumps	38
Pneumonia (lobar)	8
Poliomyelitis	2
Tuberculosis (pul.)	39
" (other forms)	9
Gonorrhea	24
Syphilis	38

WEEKLY REPORT OF COMMUNICABLE DISEASES REPORTED TO THE STATE DEPARTMENT OF HEALTH OF MAINE

FOR THE WEEK ENDING JULY 12, 1924

Chickenpox

Auburn	1
Lewiston	6
Portland	4
South Portland	1
	12

Diphtheria

Lewiston	1
Westbrook	3
	4

German Measles

Portland	1

Gonorrhea

East Boothbay	1
Kennebunk	1
Millinocket	2
Portland	8
Presque Isle	1
Rumford	2
Sanford	1
Westbrook	1
	17

Measles

Auburn	1
Bangor	3
Brownfield	2
Deer Isle	1
Kennebunkport	1
Lewiston	10
Norway	5
Portland	1
Woodstock	1
	25

Mumps

Kennebunkport	1
Madison	1
Portland	8
Rome	12
South Portland	4
Union	1
York	2
	29

Pneumonia

Brownfield	1
Caribou	1
Lewiston	2
Pittsfield	1
Portland	1
	6

Scarlet Fever

Dennistown Place	1
Lewiston	16
Old Town	1
Peru	1
Portland	2
Webster	1
	22

Septic Sore Throat

Caribou	2

Syphilis

Auburn	1
Calais	1
Portland	30
	32

Tetanus

Portland	1
Rockland	1
Skowhegan	1
	3

Typhoid Fever

Bath	10
Burnham	1
Millinocket	1
Portland	1
Sherman	1
Washington	1
York	1
	16

Tuberculosis

Portland	2

Whooping Cough

Biddeford	1
Easton	1
Lewiston	1
Pittsfield	2
Portland	7
Woodstock	4
	16

REMARKS AT THE DEDICATION OF THE NEW BUILDINGS OF THE BAPTIST HOSPITAL

BY HUGH CABOT, M. D., ANN ARBOR, MICH.

IT is a few weeks more than twenty-five years since I first began my intimate association with this hospital and on that account I think that I can see its present development from a point giving reasonable perspective.

Twenty-five years ago the hospital was being conducted in an old house, the Brown homestead. To this was added in piece-meal fashion as funds became available the semi-isolated bungalow buildings which formed the peculiar development of this institution and which have led to the present, as I believe, unique hospital structure. This period just referred to may be called the era of hospital development, and during this time hospitals have multiplied at an astonishing rate. Today in almost any part of the country one may see a new modern hospital, the structure and equipment of which has been studied by experts to the end that nothing may minimize or impede the efficiency of modern medicine in caring for the sick. In many of the great modern hospitals there are literally miles of electric wiring having for their purpose the bringing to the patient of the most delicate methods of studying and recording facts essential to accurate diagnosis. Truly science can do no more.

If a hospital be regarded as a place for the scientific study of disease, then modern buildings are at least close to the highest point of development. But it sometimse occurs to me to wonder whether this be the sole or eve nthe chief purpose that is to be sought in the building of a hospital. It would perhaps be pertinent to suggest that it is possible to confuse the study of disease with the cure of the patient. It is upon this point that I desire particularly to elaborate this afternoon, because it constitutes what I think may be properly called the Vision of the Baptist Hospital.

More than a quarter of a century ago the

trustees, many of whom are still alive, were fortunate in finding to put in charge of this then young institution a woman of extrordinary vision. She at once saw that the hospital then and for many years would be in no position to compete in point of equipment with many other hospitals with which it was surrounded. But she also had the wisdom to see that there are forces other than those of applied science which are of first-class importance in the treatment of the sick whether in body or in mind. Air, sunlight, rest, beauty and peace are forces ready to the hand of those who have the wit to seize them and yet much neglected, as I think, in modern hospital construction. Many a hospital in search of scientific efficiency tends to forget that the patient is anything but a complicated piece of machinery and yet this over-sight is capable of doing the gravest damage.

In contributng to the solution of this problem lies the unique achievement of this institution. Though progressively more and more able to offer to its patients the enormous advantages of advancing modern science, sight has never been lost of the value of environment. Quite outside the field of science, quite before the developments which have resulted in the building of this almost ideal hospital, the Baptist Hospital was offering to its patients an opportunity for recovery second to none within my experience. Today it may be said to have come to its own.

Its isolation and inaccessability have been turned to account and every advantage has been taken of its very unusual location. It has been placed upon this very carefully selected site so as to reap the full benefit of all that has been provided by nature for the healing of the

It sometimes occurs to me to wonder whether those who are hot upon the trail of modern scientific development do not sometimes fail justly to appreciate the full force of the first commandment of medicine—"Thou shalt make thy patient no worse." It is well to remember that no physician ever cures his patient, but only assists him in healing himself. We are too prone to neglect the healing powers of nature in our interest in the healing appliances of science. It is by no means easy, perhaps not always possible, to decide the precise point to which modern science can be applied wholly to the advantage of the patient, but we can at least avoid insult whether physical or spiritual to those who are handicapped by injury or disease. Yet many a modern hospital is constructed in utter disregard of this self-evident truth. From start to finish the patient is met with unfavorable suggestions of smell, sight and sound quite capable of retarding recovery or, perhaps worse, implanting in his consciousness suggestions which may do lasting damage.

sick. It will be possible here to live quite shut off from the unsavory atmosphere of a hospital, to enjoy the advantages of light, air and beauty to the fullest. It should be observed that attention has been paid to withdrawing the ordinary machinery of the hospital from the sight and hearing of the patient. The surgical workshops, more commonly referred to in resounding phrase as operating theatres, have been kept out of the way, these work-shops of the surgeon where miracles may be performed, but in which it is all too easy to implant in patients' minds suggestions of tragedy and death which are astonishingly permanent. Every effort has been made to remove even the "hospital" smells never noticed by the physician and never overlooked by the patient from their normal obtrusiveness.

This hospital may be regarded as a most interesting development in hospital construction from which it will be possible to estimate to what extent we have neglected the forces of nature and relied too implicitly upon the science

rather than the art of medicine. This is, furthermore, a timely demonstration for, in these days when knowledge particularly of new developments in science spreads with great rapidity and is unhandicapped by distance, we seem to me to be in the gravest danger of over-estimating the part played by science and of pushing into the background forces which may perhaps probably be described as spiritual which have a very far-reaching effect on the ultimate result to the individual of his experience in the field of disease.

To quote a line from Kipling which seems to me apposite in this connection: "What I ha' seen * * * * leave me no doot for the machine: but what about the man?"

ROCKEFELLER INSTITUTE APPOINTMENTS

July 3, 1924.

THE Board of Scientific Directors of The Rockefeller Institute for Medical Research announces the election of Dr. Francis Gilman Blake as a member of the Board of Scientific Directors to succeed Dr. Hermann M. Biggs, deceased.

The following promotions and appointments are announced:

PROMOTIONS

Associate Member to Member: Dr. James B. Murphy, Dr. John H. Northrop.

Associate to Associate Member: Dr. Thomas M. Rivers.

Fellow to Assistant: Miss Gladys Bryant, Dr. Charles Korb, Miss Dorothy Loomis, Dr. Elmer L. Straub.

Dr. William S. Tillett has been appointed Resident Physician at the Hospital.

NEW APPOINTMENTS

Assistants: Dr. Douglas Boyd, Dr. Clifford L. Derick, Dr. Louis A. Julianelle, Dr. Ann G. Kuttner, Dr. John F. McIntosh, Mr. Bernard J. C. Vander Hoeven.

Fellow: Dr. David Davidson.

Dr. Paul E. Howe, hitherto an Associate in the Department of Animal Pathology, has accepted an appointment with the Division of Animal Husbandry, Bureau of Animal Industry, U. S. Department of Agriculture, in charge of Nutrition Investigations.

Dr. George R. Brow, hitherto an Assistant in the Department of the Hospital, has accepted an appointment as research assistant at the University College Hospital, London, under Sir Thomas Lewis.

Dr. Geoffry C. Linder, hitherto an Assistant in the Department of the Hospital, has accepted an appointment at St. Bartholomew's Hospital, London, under Professor Francis R. Fraser.

Dr. Hugh J. Morgan, hitherto an Assistant and Resident Physician at the Hospital, has accepted an appointment as Associate Professor of Medicine at Vanderbilt Universty.

Dr. James M. Neill, hitherto an Assistant in the Department of the Hospital, has accepted an appointment as Associate Professor of Bacteriology at Vanderbilt University.

Dr. Harald A. Salvesen, hitherto an Assistant in the Department of the Hospital, has accepted an appointment as Chief of Clinic at the Physiologic Institute, University of Christiania, Norway.

THE ANNUAL MEETING OF THE ASSOCIATED BOARDS OF HEALTH OF THE SOUTHEASTERN DISTRICT OF MASSACHUSETTS

THE annual meeting of the Associated Boards of Health of the Southeastern Health District of Massachusetts was held in City Hall, Fairhaven, on Wednesday, July 9, with representatives present from Acushnet, Attleboro, Barnstable, Berkeley, Brewster, Bourne, Dennis, Fairhaven, Falmouth, Marion, Mashpee, Middleboro, New Bedford, Wareham and Westport. The election of officers resulted in the following list to serve the coming year; President, Walter K. Perry of Marion; Vice-President, W. Fred Delano of Fairhaven and William Crowell of Dennis; Secretary-Treasurer, G. W. Hallet of Barnstable; Executive Committee, Dr. C. W. Milliken of New Bedford, Harry B. Albro of Falmouth and Fred E. Wellington of Acushnet.

The speakers were Herman C. Lythgoe of the Mass. Department of Public Health, Division of Foods and Drugs, who outlined the new sanitary law of the State and Dr. John T. Pinckney, Chief of Clinics of the Division of Tuberculosis, also of the State Department of Public Health, who spoke concerning the State ten-year program on tuberculosis. There was much close discussion following the papers. The morning session concluded with resolutions on the death of Capt. Anthony Bryer of Brewster.

The discussions were continued during a Shore Dinner at Fort Phoenix and the afternoon session took up in round table fashion a large number of matters of health administration technique.

NOTICE

BERKSHIRE DISTRICT MEETING

DR. JOHN B. DEAVER of Philadelphia will speak before the Berkshire District Medical Society on July 31st, at the Hotel Aspinwall, Lenox, on the subject, "The Upper Abdominal Diseases." The society extends a cordial invitation to physicians. Dinner will be served at 6.30. The speaking will follow.